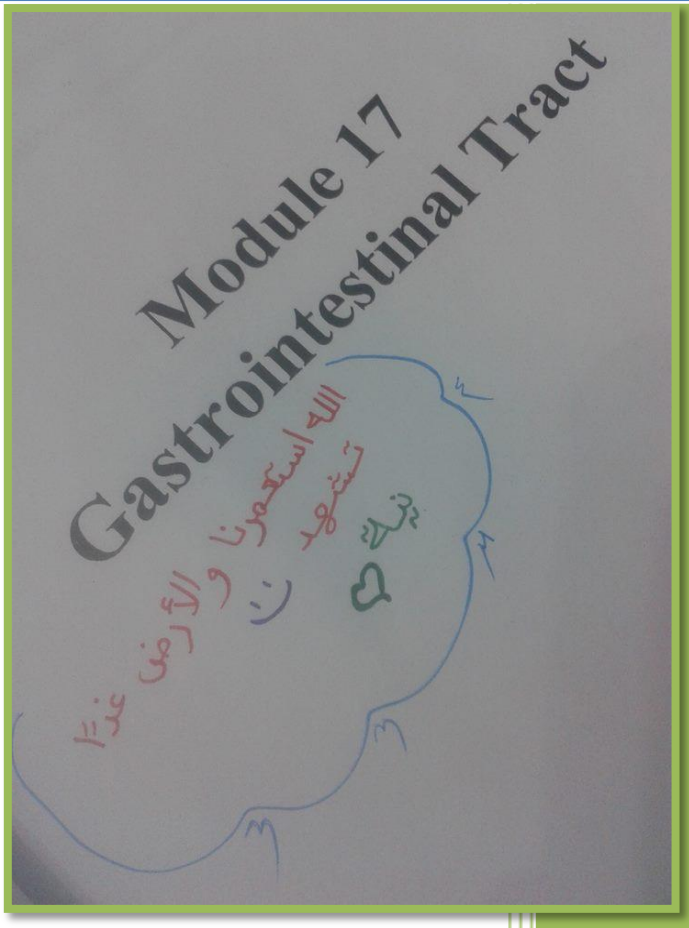
ASM 2019 Team

GIT answered essay Questions



1- Physiology ^_^

استعن بالله ولا تعجز ،،

طول ما فى نفس ،، وطول ما فى ولو دقيقة ،، تقدر تكتب نهاية جديدة

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بسم الله

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1. Describe the types of intestinal movements.

There are mainly 3 types of movements: Segmenting (mixing), peristaltic (propulsive) and tonic contractions.

1. ***Segmenting movements (mixing contractions)***

- Segmentation of the small intestine occurs in response to distension with chime.

- They are ring like contractions dividing a loop of the intestine into equal portions, giving the appearance of a chain of sausages. Then, these contractions relax and a new set of contractions begin in the previously relaxed points.

- Segmentation
  >>> **mixes** the intestinal contents with the digestive juices,
  >>> **moves** the chyme to and fro and **increases its exposure** to the mucosal surface.
  >>> it **slows transit** in the small intestine to permit longer contact of the chyme with the enterocytes and fosters absorption

- The maximum frequency of the segmentation contractions in the small intestine is determined by the frequency of *electrical slow waves* in the intestinal wall, which is the basic electrical rhythm. So, they occur at a rate of 12/minute in the duodenum and decrease to 8/minute in the terminal ileum.

- Although slow waves in the smooth muscle cause the segmentation contractions, these contractions are ineffective without background excitation mainly from the myenteric nerve plexus. These movements occur independent of extrinsic innervation (myogenic in nature).

2. *Peristaltic movements (propulsive movements)*

- Peristalsis is a reflex response that is initiated when the gut wall is stretched by the contents of the lumen, and it occurs in all parts of the gastrointestinal tract from the esophagus to the rectum.

- The stretch of duodenal wall initiates a circular muscle contraction behind the stimulus and an area of receptive relaxation in front of it.

- The wave of contraction then moves in an oral-to-caudal direction, propelling the contents of the lumen forward.

- Peristalsis is an excellent example of the integrated activity of the enteric nervous system. It appears that local stretch releases serotonin, which activates the myenteric plexus. Then, impulses pass in a retrograde direction in this plexus to activate the release of substance P and acetylcholine, causing circular smooth muscle contraction. At the same time, impulses passing in an anterograde direction activate the secretion of NO, VIP, and adenosine triphosphate (ATP), producing the relaxation ahead of the stimulus.

- In addition to stretch of duodenum, the peristaltic activity of the small intestine could be initiated by gastroenteric reflex: Distention

of the stomach excites some receptors and this reflex is carried out by the myenteric plexus from the stomach down along the wall of the small intestine.

- Peristalsis promotes propulsion of food through the small intestine at a velocity of 0.5 to 2 cm/second, it is much slower in the distal than in the proximal parts. They die out after few centimeters. The movement of food along the small intestine normally averages only 1 cm/min. This means that 3 to 5 hours are required for passage of chyme from the pylorus to the ileocecal valve

- Peristaltic movements lead to forward movement of food toward the ileocecal valve and spread out of chyme along the intestinal mucosa.

N.B. Segmenting movements have some propulsive effect, and peristalsis has some mixing effect.

4. *Antiperistalsis*

This is peristalsis in opposite direction, i.e.
a) Between duodenum and stomach: to allow time for neutralization.
b) Between ileum and caecum; to allow time for absorption.

5. *MMC*

In the period between meals, the intestine is relatively quiescent, but every 90 min or so it is swept through by a large peristaltic wave triggered by the hormone motilin.

N.B. **Peristaltic rush:** is a powerful series of peristaltic waves that travel along the small intestine in a few minutes. It occurs in some infectious diseases, i.e. it is not present in normal conditions. Peristaltic rush takes the intestinal contents toward the colon to get rid of any distention or irritation

2. Explain the function of saliva.

1. **It moistens** food to facilitate its swallowing.

2. **It cleans** the mouth by:
   a. Mechanical wash of the food residues and the shed epithelial cells which prevents putrefaction.
   b. Antibacterial action by lysozyme.
   c. The bactericidal effect of thiocyanate ions.
   d. Saliva contains protein antibodies that can destroy the bacteria in the oral cavity.

3. **It facilitates speech** by moistening of the oral cavity which includes soft palate, tongue and lips.

4. It has a **role in digestion of starch** by salivary amylase. Digestion of starch is completed by pancreatic amylase.

5. It dissolves some food materials to facilitate its stimulating effect on **taste** buds (helps taste sensation).

6. It has a role in water balance because dryness of the buccal and pharyngeal mucosa initiates the sensation of **thirst**.

7. It has a **buffering** action via its contents of bicarbonates, phosphates, and mucin, to protect the buccal cavity from excessive acids or alkalis. Excessive alkalis lead to precipitation of calcium salts around the teeth which forms with organic matter hard concretions called tarter, bacteria flourish underneath the Tarter leading to chronic inflammation of the gums. Excessive acids dissolve enamel and dentine of teeth after prolonged exposure.

8. It protects against irritating substances and neutralizes of excessively cold or hot food.

9. It helps in body **temperature regulation** in animals with no sweat glands (e.g. dogs) by evaporation of saliva.

10. It excretes iodides and mercury which may produce inflammation of buccal mucosa (stomatitis). Also, in lead poisoning a blue line formed at the margins of teeth and gums. Urea is excreted in saliva in renal diseases and some little glucose may be also excreted in diabetes mellitus. This excretory process is not very effective because these materials are swallowed again.

Explain the factors affecting intestinal absorption.

Any factor which affects the metabolic activity of the intestinal mucosa reflects itself on intestinal absorption. Also physico-chemical factors affect it.

1. Viability of the intestinal mucosa
An adequate blood flow, oxygen supply, and the general metabolic activity of the intestinal mucosa greatly affect the rate of absorption.

2. Lymph flow
Obstruction of the thoracic duct hinders fat absorption but does not abolish it completely due to the presence of other lymphatic collaterals.

3. Proper digestion
Completely digested food is easily absorbed; improper digestion is accompanied by malabsorption

4. Intestinal movements
As mentioned before, intestinal movements and the movements of the villi greatly enhance the rate of absorption. However, excessive peristalsis

hinders absorption due to hurrying the intestinal contents as in case of diarrhea.

5. Some physical and chemical factors

a. Hypertonic solutions are absorbed at a slower rate than hypotonic or isotonic solutions.
b. Crystalloids are absorbed according to their concentration gradients between the intestinal lumen and plasma.
c. Increased intra-intestinal pressure increases the rate of absorption.
d. Solubility: solubility of the substances is necessary for their proper absorption, for example bile salts facilitate absorption of fat and fat soluble vitamins by dissolving them. Also, absorption of calcium is increased when the reaction of the intestinal contents is acidic, because the solubility product is increased in this acidic medium.

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3. Discuss the hormonal regulation of gastric secretion.

Through gastric and intestinal phases.

a) Gastric phase

In this phase gastric secretion occurs while the food is in the stomach itself and in contact with gastric mucosa.

››› is due to release of gastrin hormone, which is a polypeptide, released by the antral part of the gastric mucosa (gastrin cells or G cells) in response to:

1. Distension of the stomach by food.
2. Certain substances called secretagogues, such as vegetables extractives, digestive products of proteins, alcohol and caffeine.
3. Vagal stimulation.

stimulation of the gastric mucosa as previous >>> >>> discharging impulses from the stomach wall >>> to the cell bodies of the local nerve plexuses >>> which in turn transmit efferent impulses to the G cells >>> causing them to secrete **gastrin**.

The neurotransmitter (from vagal fibers) at the G cells seems to be bombesin (=gastrinreleasing peptide = GRP) and not acetylcholine.

(الحتة دى مهمة اعرف ان ال neurotransmitter اللى بيطلع يأثر على ال G cell او بمعنى اخر ال neurotransmitter اللى غرضه انه يطلع ال gastrin اسمه gastrin releasing peptide او GRP او الاسم الاخير والاهم bombesin)

Gastrin goes to the blood, and is carried to oxyntic glands in the body of the stomach and peptic cells via their arterial blood supply. Any factor which blocks this reflex stops the release of gastrin.

b) Intestinal phase

When the products of gastric digestion enter the duodenum, they stimulate secretion of intestinal gastrin from the duodenal mucosa in response to distension or chemical stimuli (the same as in the gastric phase). Intestinal gastrin reaches the stomach via its blood supply and stimulates the gastric glands in the fundus and body of the stomach to secrete a strongly acidic juice, rich in enzymes. Also other hormones are secreted to inhibit gastric secretion and emptying e,g. secretin, Cholecystokinin (CCK), neurotensin, Gastroinhibitory peptide (GIP), ...

Secretin is secreted by the duodenal mucosa in response to acidification of the bulb. In other words, the stimulus for its secretion is the evacuation of an acidicchyme before adequate mixing and buffering of the acid.
Cholecystokinin is secreted by duodenal mucosa in response to contact with fat (or what is literally called a heavy meal).
It is clear, from the above, that the distal parts of the gut have some command on the stomach. In other words, the stomach is controlled both from above (the brain) and from below (the small intestine).

4. Discuss phases of deglutition and its nervous pathway

Deglutition is divided into 3 phases to facilitate its description but really once the first phase is started the others follow without an interval in between.

The first (voluntary) phase
Food in the mouth is ―voluntarily‖ squeezed or rolled posteriorly into the pharynx by pressure of the tongue upward and backward against the palate.

The second (pharyngeal) phase

- It is involuntary reflex and starts by the passage of food through the pharynx.
- It is a very rapid phase which takes about 1-2 seconds. The constrictor muscles of the pharynx contract, with receptive relaxation of the upper oesophageal sphincter.
- The posterior pillars of the fauces approximate to shut off the mouth cavity from the pharynx and prevent the back passage of food to it.

Important protective reflexes take place to prevent the passage of food into the respiratory openings:

a) Elevation of the soft palate to shut off the posterior nares and prevent the passage of food into the nasal cavity.
b) Elevation of the larynx and closure of its opening (glottis) by the epiglottis and posterior part of the tongue and approximation of the vocal cords to guard against the passage of food into the air ways.
c) Inhibition of respiration by a reflex mechanism (reflex apnea).

In anesthesia, cough and swallowing centers are depressed and the protective reflexes do not occur. Secretion or vomitus may accumulate in the pharynx and enter the trachea leading to choking.

The third (oesophageal) phase

In this involuntary phase, food passes along the oesophagus to the stomach by oesophageal peristaltic contractions (described above). The movements in the upper third of the oesophagus are rapid while those in the lower third are slow, because the muscles of the upper third are striated and supplied by the vagus nerve while in its lower third are plain and depend on local plexus of Auerbach. The middle third contains both types of muscles. This phase is helped by mucin and gravity in erect position.

(A) The tongue pushes the food bolus to the back of the mouth.
(B) Soft palate elevates to prevent food from entering the nasal passages.
(C) The epiglottis covers the glottis to prevent food from entering the trachea and the upper oesophageal sphincter relaxes.
(D) Food descends into the oesophagus

Nervous pathways in deglutition (Figure 4-4)
The reflexes of the involuntary phases are mediated by:
1. **Afferents:** start from receptors that discharge impulses from the upper pharynx along the 5th, 9th, 10th cranial nerves.
2. **Centre:** is the deglutition centre in the medulla oblongata.
3. **Efferents** along the 5th cranial nerve to mylohyoid muscles, the 9 th,10th and
11th cranial nerves to the pharyngeal and oesophageal muscles and the 12th cranial nerve to the tongue muscles

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Mention the function of bile salts.

1. ***Digestion of fat:***

Bile salts have no direct digestive effects, but help fat digestion by:

- ***Emulsifying*** or ***detergent function***. Emulsification decreases the surface tension of the particles and allows a good surface area to be exposed to the action of pancreatic lipase.

- Bile salts also activate lipase enzyme.

2. ***Absorption of fat:***
   - Bile salts tend to form "**micelles**". The micelle contains fatty acids, monoglycerides, phospholipids and cholesterol Thus; it solubilizes the lipids and provides a mechanism for their transport to the enterocytes. The micelles move down their concentration gradient through the brush border of the intestinal mucosal cells, where they are absorbed. Then, the lipids diffuse out of the micelles, and the absorbed bile salts are transported back to the liver in the portal vein and re-excreted in the bile (enterohepatic circulation). Bile salts have the ability to dissolve the insoluble fatty acids in water, thus the fatty acids formed are removed from medium and new molecules of neutral fat are exposed to the action of pancreatic lipase.

   - Bile salts facilitate the absorption of fat-soluble vitamins (D, E, K) and carotenes. Absorption of fatty acids also helps indirectly the absorption of calcium and iron by prevention of their precipitation in an insoluble form.

3. ***Solvent action:*** Because bile salts prevent the precipitation of cholesterol and fatty acids by keeping them in solution, they guard against gall stones formation.

4. ***Choleretic function:*** Bile salts are the best choleretics (stimulants for bile secretion by liver cells). Absorption of any amount of the secreted bile salts in the intestinal mucosa passes via the portal circulation to the liver, leading to stimulation of bile secretion.

5. ***Stimulation of peristalsis:*** Bile salts stimulate the motility of the intestine and colon and prevent constipation.

6. ***Anti-putrefactive action:*** Bile salts have no direct antiseptic effect, but they prevent putrefaction by absorption of fats. In their absence,

undigested fat covers the protein particles and hinders their digestion. The undigested protein particles in the large intestine are attacked by bacteria leading to putrefaction.

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1. Describe the formation of HCL and its functions

The most probable mechanism of HCl secretion is as follows

1- Parietal cells exert electrochemical work to **dissociate a molecule of water into H^+ and OH^-.** Active transport of H^+ across the membrane of the parietal cells to the gastric lumen occurs (proton pump). The energy for this active transport is derived from aerobic glycolysis, since acid secretion ceases under anaerobic conditions. Two molecules of H+ are secreted for each molecule of O_2 consumed.

2- CO_2 from the blood enters the parietal cells where it reacts with water to form carbonic acid (H_2CO_3), catalyzed by carbonic anhydrase enzyme. Dissociation of H_2CO_3 into H^+ and HCO_3^- occurs. H^+ combines with the OH^- produced in the first step forming H_2O.

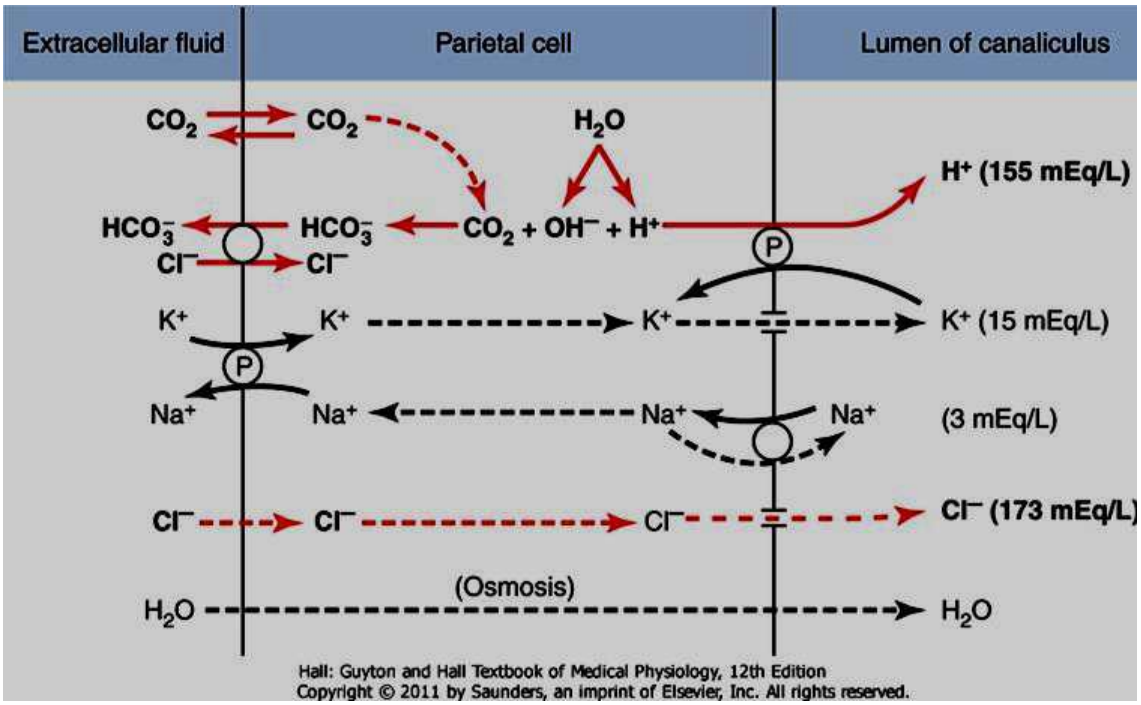
$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow H^+ + HCO_3$

Secretion of HCl is directly proportional to the CO_2 tension of blood: in hyperventilation, HCl formation is depressed. Diamox, a carbonic anhydrase inhibitor also depresses gastric secretion of HCI.

3- Bicarbonate (HCO_3^-) ions pass from parietal cells to the blood in exchange for chloride ions (Cl^-) that enter the cell. Cl^- ions are secreted by the parietal cells at the luminal side of the gastric mucosa by an active process against a relatively high concentration gradient and are coupled with the H^+ ions produced in the first reaction, giving a strong solution of hydrochloric acid (HCl) in the canaliculus. The HCl is then secreted outward through the open end of the canaliculus into the lumen of the gland.

Blood coming from the stomach is alkaline, and has a high HCO_3 content.

When gastric acid secretion is elevated after a meal, the pH of the systemic blood rises and alkaline urine is excreted. This is the probable explanation of the high pH of urine excreted after a meal, the "postprandial alkaline tide".



Functions of HCl

1. Activates the inactive proteolytic enzyme pepsinogen to the active form, pepsin, and gives the optimum pH needed for its action.
2. Has an antibacterial action by killing microorganisms so prevents their entry into the duodenum and biliary tract.
3. Facilitates the absorption of calcium and iron.
4. Helps the hydrolysis of some food materials as disaccharides.
5. Acid chyme stimulates the release of secretin hormone from the duodenum and thus initiates the entero-gastric reflex which regulates the rate of emptying of the stomach.

2. Discuss the regulation of exocrine pancreatic secretion.

By both nervous and hormonal mechanisms, but hormonal regulation is more important.

1. Nervous regulation

Nervous type of secretion occurs by: conditioned and unconditioned reflexes. The same nervous signals from the brain that cause secretion in the stomach also cause acetylcholine release by the vagal nerve endings in the pancreas. It stimulates pancreatic acinar cells to cause discharge of zymogen granules. ***Nervous type of secretion is small in amount, rich in enzymes but deficient in alkali and water.***

*(واكتب انت بأسلوبك .. **same signals** دى يقصد بيها ان نفس حالات ال **conditioned** زى تشوف او تسمع ،، وحاجات ال **unconditioned** زى ان الاكل يبدأ ف الطريق من أول ال **mouth** كله يشتغل ،، فيؤدى لل **brain** اللى جاى من ال **vagus** بال **mediated** اللى ،، **reflexes** اللى هو **secretion** من ال **acini** مش **water** و **alkali** **enzymes**)*

2.Hormonal regulation

Gastric phase ››› gastrin which increases pancreatic juice rich in enzymes

Intestinal phase ›› **secretin**, ***CCK*** and **VIP**.

a.Secretin : Secretin is secreted by S cells that are located in mucosa of the upper portion of the small intestine It is present in an inactive form, pro-secretin. The entry of acid chyme to the duodenum, leads to its activation ‹‹leads to

››› copious secretion of a very alkaline pancreatic juice poor in enzymes. It is believed to act on duct cells ›› secrete water and bicarbonate.
The release of secretin by acid is an example of negative feedback control.

Secretin causes alkaline pancreatic juice to flood into the duodenum, neutralizing the acid from the stomach and thus inhibiting further secretion of the
hormone.

b.*CCK*: secreted from the I cells at the mucosa of the upper small intestine in presence of digestive products or proteins, and fats.

››› Cholecystokinin evokes pancreatic secretion rich in enzymes and poor in water and bicarbonates, which is similar to the effect of vagal stimulation.

Because the bile and pancreatic juice that enter the duodenum in response to CCK further catalyze the digestion of more protein and fat. The products of this digestion stimulate further CCK secretion, a sort of positive feedback operates in the control of the secretion of this hormone. However, the positive feedback is terminated when the products of digestion move on to the lower portions of the gastrointestinal tract.

c.Vasoactive intestinal peptide (VIP)

It stimulates secretion of water and bicarbonate from pancreas, but it is weaker
than secretin.

3. Describe unconditioned reflexes controlling salivary secretion

Stimulus:

- Food, acids or alkalis are introduced into the mouth, stimulating the buccal mucosa **mechanically** or **chemically**.
- irritation of buccal cavity by moving the tongue inside the mouth or during speech

Receptors

1. The four types of taste receptors in the tongue namely salt, acid, sweet, and bitter receptors.
2. Thermoreceptors and pain receptors in the buccal cavity.
3. Pharyngeal and oesophageal receptors.

***Afferent*:**

- Impulses for taste sensation from the **anterior two third**s of the tongue are conducted by the **chorda tympani** branch of the facial nerve

- **posterior third** of the tongue and pharynx impulses for taste sensations are carried by the **glossopharyngeal** and **vagus** nerves.

- Impulses from **mechanical** stimuli and **common sensations** (touch, pain, warm and cold) are conducted by **the lingual nerve, palatine branch of the trigeminal nerve and pharyngeal branch of the vagus nerve**.

Centre:

- Parasympathetic: salivatory centers in medulla oblongata.
- Sympathetic: lateral horn cells in the first and second thoracic segments of the spinal cord.

Efferent: along the autonomic nerves supplying the salivary glands (Figure 5).

Effectors: Salivary glands

Response: Salivary secretion

– Sympathetic: produces viscid organic secretion.

– Parasympathetic: produces watery secretion.

4. Discuss factors affecting gastric emptying

I) Gastric factors

a) Volume of the gastric contents

Distension of the stomach incrases the rate of emptying of the stomach through nervous mechanism. The rate is directly proportional to the square root of food volume in the stomach.

b) Effect of gastrin hormone on stomach emptying

stretch of the stomach wall and the presence of certain types of food in the stomach release gastrin hormone, which stimulates both gastric secretion and motility. It increases the activity of the pyloric pump and relaxes the pyloric sphincter, i.e. it enhances emptying of the stomach.

II) Duodenal factors

Feedback mechanism from the duodenum is very important to slow down the rate of evacuation of the stomach when the small intestine is already filled, this is obtained through:

- ***Enterogastric reflex***

When chyme enters the duodenum reflex inhibition of antral peristalsis associated with contraction of the pylorus occurs (enterogastic reflex) this is

medited by three ways; as a local enteric reflex, a prevertebral ganglionic and vago-vagal reflex. This is the most important factor in controlling the rate of emptying of the stomach.

The most important stimuli for enterogastric reflex are the irritants and acids in the duodenal chyme. Acids are prevented from release in the duodenum to allow time for neutralization by pancreatic and other secretions.

- ***Hormonal factors***

Cholecystokinin is released from the jejunal mucosa in response to fatty chyme. This hormone blocks gastric motility by competitive inhibition of gastrin hormone.

Secretin is secreted from the duodenum in response to acid chyme and it decreases gastric motility.

Gastroinhibitory peptide (GIP) is released from the upper small intestine in response to fat and to a lesser extent in response to carbohydrates. It inhibits gastric acid secretion and motility. Its main function is probably to stimulate the release of insulin from the pancreas after a meal.

Somatostatin is secreted in the enteric plexus of the GIT. It inhibits the secretion of gastrin and inhibits gastric emptying.

Vasoactive intestinal peptide (VIP) mainly decreases HCl secretion and motility.

Ill) Some other factors which affect gastric emptying:

a) Chemical agents

Cholinergic drugs, sodium bicarbonate, coffee, alcohol, and vitamin B complex members are stimulants to gastric movements. Adrenergic drugs, cholinergic blockers (as atropine), smoking, and bacterial toxins depress stomach emptying.

b) Emotions

In anxiety and anger, gastric motility and secretion are greatly increased, while fear, pain, depression, sadness, and muscular exercise depress them.

Persistent excitation leads to peptic ulceration.

c) Consistency of food

Emptying of fluids is more rapid than semisolids, which in turn are emptied faster than solids. Solid food takes a longer time until it is converted to "chyme" by proper mixing and partial digestion by gastric juice.

d) Automatic stimulation

Sympathetic inhibits motility while parasympathetic increases motility.

e) Type of food

Proteins (particularly meat) stimulate the release of gastrin→ increase gastric motility and inhibit pyloric sphincter→faster emptying. Fat (heavy meal) inhibits motility and emptying. The entry of fats and fatty acids in the duodenum release some duodenal hormones that delay gastric emptying.

They decrease gastric motility and increase the tone of pyloric sphincter.

Discuss functions of bile salts >> mokrar imp.
Func. Of saliva >> mokarar imp.

Mention protective reflexes during deglutition (as previous)

Important protective reflexes take place to prevent the passage of food into the respiratory openings:

a) Elevation of the soft palate to shut off the posterior nares and prevent the passage of food into the nasal cavity.
b) Elevation of the larynx and closure of its opening (glottis) by the epiglottis and posterior part of the tongue and approximation of the vocal cords to guard against the passage of food into the air ways.
c) Inhibition of respiration by a reflex mechanism (reflex apnea).

Types of intestinal motility >>mokrar
(:D: ودى اشياء مطمئنةD)
Reg. of pancreatic secretion >> :D
Factors affecting absorption >> :D
Mention 4 func. Of saliva >> :D

Mention 3 types of gastric motility

a) Mixing movements: Squirt the stomach contents back into the gastric lumen (retropulsion).
b) Propulsive movements: Squirt the contents through the pylorus into the duodenum. The pyloric aperture acts as a sieve that allows only small particlesto pass through.
c) Grinding movements: The pyloric antrum and canal remain an isolated cavity with strong contractions in their wall to press the contents.
d) Migrating motor complex (MMC)
During the inter-digestive period any food left in the stomach is removed by the MMC which is a peristaltic wave that begins within the esophagus and travels through the entire gastrointestinal tract. This peristaltic wave occurs every 60-90 min

during the inter-digestive period. The hormone **motilin** which is released fromendocrine cells within the epithelium of the small intestine increases the strength of MMC.

Functions of the gall bladder

1. Storage of bile

Bile flows into the duodenum only after ingestion of food, in between the meals the sphincter of Oddi is closed, and bile flows back to the cystic duct to be stored in the gall bladder.

2. Concentration and acidification of bile

The total daily secretion of bile is about 500 ml, while the maximum volume of gall bladder is only 40-70 ml. Nevertheless, as much as 12 hours bile secretion can be stored because its water, sodium chloride and most other small molecular weight electrolytes are absorbed by the gall bladder mucosa. The other bile constituents are concentrated. Bile can be concentrated up to 10 or 12 times. Acidification is done by absorption of its NaHCO3 content, which prevents the precipitation of insoluble calcium salts, and formation of gall stones. Also acidity has bactericidal effect.

3. Secretion of white bile

Gall bladder and bile duct mucosa secrete mucoid like material which protects them from any irritant chemical products.

4. Buffering of biliary pressure

The gall bladder equalizes the pressure in the biliary system. By storing bile, it prevents any increase in pressure in biliary passages. This enables the liver to secrete because hepatic cells cannot secrete against high pressure.

5. Evacuation of bile when needed

Hepatic cells secrete bile at a low pressure, about 7 cm H_2O. The tonic contraction of the sphincter of Oddi is equivalent to 30 cm H_20 when it is closed
(in between the meals). After ingestion of food there is partial relaxation of the sphincter with a tone of about 10 cm H_2O, synchronous with contraction of the gallbladder with a pressure equal to 20 cm H2O. In this case the bile flows from the gall bladder together with the fresh bile coming from the liver to the duodenum.

Discuss inborn control of salivary secretion>>=unconditioned :D

Discuss types of int. motility >> :D

Discuss func. Of the liver

I. Digestive function: formation and secretion of bile

II. Non digestive functions

1. It acts as a blood reservoir and as a mean of transferring blood from portal to systemic circulation. A normal volume of about 350 ml of blood is found in the organ. An additional liter may be stored in the organ and transferred to systemic circulation.
2. The liver serves as a filter between the blood coming from the gastrointestinal tract and the blood in the rest of the body through the activity of phagocytic reticulo-endothelial cells (Kupffer cells), found in the sinusoids.
3. Production of red blood cells is a normal function of fetal liver.
4. Destruction of aged red blood cells by the activity of kupffer cells.
5. Excretion of bile pigments.
6. Detoxification function: The liver inactivates toxins, hormones and steroids that might be harmful to the body by oxidation, hydroxylation, and other reactions mediated by the large number of cytochrome P450 enzymes expressed in hepatocytes. Ultimately, metabolites are secreted
into the bile for elimination via the gastrointestinal tract.
7. Metabolic functions: The liver plays key roles in carbohydrate, fat and protein metabolism. For example, it plays a major role in maintaining the stability of blood glucose levels in the postprandial period, removing excess glucose from the blood and returning it as needed—the so-called glucose buffer function of the liver. In addition, the liver preserves cholesterol homeostasis by synthesizing this molecule and also converting excess cholesterol to bile acids.
8. Storage of iron, vitamins.
9. Endocrine function: synthesis of erythropoietin, somatomedins, etc.
10. Helps in blood clotting mechanism (synthesis of clotting factors)
11. Synthesis of plasma proteins such as acute phase proteins, albumin, hormone binding proteins, etc

Done

سيبتلك الاسئلة المتكررة مع كل مرة تتكرر بكتبها عشان تتطمن :D ان شاء الله

على فكرة كدة الفسيولوجى اضمن بيها درجات ذاكر الكام سؤال اللي بيتكرروا ^_^

2- Histology

VERY IMPORTANT:

Familiarize yourself with the photos and diagrams in the book. Old exams want you to draw. Recent exams just want you to recognize a photomicrograph or a diagram with all its labels.

1. Give a brief account with an illustrated diagram of a mixed salivary acinus.

- They are mainly present in the submandibular and sublingual glands.
- They are formed mainly by mucous cells with some serous cells interspersed among them. B oth cells pour their secretion directly into the lumen.
- In routine preparations, due to swelling of mucous cells and compression of the serous o nes, mucous cells appear as if surrounded by deeply stained caps of serous cells called the crescents of Gianuzzi or the serous demilunes.

2. Mention five different histological features between a filiform and a circumvallate papilla.

	Filiform	**Circumvallate**
Number	Most numerous (Fili shabah " =Fill "يملئ3shan kda hya kteer)	6-14
Shape	Long,Slinder,Conical –inclinationin an tro-posterior direction "The Longest" (shabah "L"m2lop)	Large,triangular,flattened upper sur face &narrow base(the largest) visi ble by naked eye -Has trench where Von Ebner's glan ds pour their secretion into
Distribution	Rows parallel to gustatory line over a nt.2/3 of the dorsum of the tongue	Aligned just in front of the gustator y line

Epithelium	**Keratinized** str. Squ.epi. split at the tip (shed with saliva)	Non-keratinized str. Squ.epi.
Secondry Papillae	Very poorly developed	Short
Taste Buds	Lacking	Numerous ,confined to the sides of the papillae
Notes	In GIT disturbance & fever accumulation of keratin occur & mixed with bacteria forming grey film(coated tongue)	**Von Ebners Glands:**serous glands,at the buttom of the trench secrete lipase enzyme & dissolving food particles to be tasted

(Thanks to **Yousra Kamal** and her file ☺ http://bit.ly/1GTco6P)

*Similar Question from **2014**:*

Enumerate the lingual papillae that are present in human. Describe the structure of the largest papillae. (1.5 marks)

3. Tabulate the differences between the muscosa of the stomach fundus and that of the ileum.

	Stomach fundus	**Ileum**
Surface Epithelium	short narrow gastric pits in 1/5 of height. -simple columnar epi. (surface mucous cell)	cover the villi & lines the crypts of Lieberkuhn. Each villus is formed of : a) central core of loose C.T. rich in lymphoid cells. Contain : Capillary loops, a blindly ended lymphatic channels (lacteal) & few smooth muscles b) villous epithelium:

		Absorptive columnar cells and M cells
Lamina propria	Scant loose highly vascularized C .T. has gastric glands & lenticulat e nodules (aggregates of lympho cytes near the bases of the gland s)	contains Peyer's patches (aggregation of lymphoid nod ules having a pale germinal ce nter occupied by lymphoblast and antibody produ
Muscularis mucosa	Thin layers of inner circular & ou ter longitudinal layers	Inner circular and outer longitudinal underlying the bottom of the Crypt

5. Mention the characteristic features of the submandibular gland.

- The capsule and septa are moderately thick.
- Fat cells are present between the acini but less numerous than in the parotid gland.
- The secretory acini are mainly of the serous types (80%) and only about 20% are of the mucous type.
- The striated secretory ducts are more prominent than in the parotid gland. They for m about 80% of the length of the intralobular ducts, while the remaining 20% are formed by the intercalated segments.

6. Describe the histological features of the space of Disse.

- It is a narrow perisinusoidal space that exists between the endothelial lining of the sin usoids and the hepatocytes.
- It contains:

1. Reticular fibers traversing the space and forming a support to the wall of sinusoids prevent ing their collapse.

2. Microvilli projecting from the hepatocytes. The space contains plasma filtered from the si nusoidal blood that constantly baths the hepatocyte microvilli accelerating the exchange o f metabolites between liver cells and blood stream.

3. Lipocytes or Ito cells which are fat storing cells. They are stellate in shape, present in-b etween hepatocytes and send many supporting processes into the space of Disse. They h ave a role in vitamin A metabolism, transport and storage. In chronic inflammation, these cells differentiate into fibroblast like cells and deposit collagen in the perisinusoidal space c ausing liver fibrosis.

7. Describe the histological features increasing the absorptive surface area of different parts of the small intestine and how does it adapt to that function.

1. Grossly, the lining of the small intestine shows a series of permanent folds, the **plicae circularis (valves of Kerckring)**, consisting of the mucosa and submucosa and having a se milunar, circular or spiral form. These plicae are most developed in the jejunum.

2. **Intestinal villi**: The villi are most numerous in the duodenum and the proximal jejunum. The y are relatively short, broad and leaf-like in shape in the duodenum; they are the longest, thinn er and finger-like in the jejunum; whereas in the ileum they are finger-like. Accordingly food abs orption is maximal in the jejunum, followed by the duodenum and is the least in the ileum.

3. **Microvilli**: modification in the apical plasmalemma of the epithelial cells covering the intestin al villi.

8. Give an account on: liver acinus.

- **This is the functional unit of the liver.**
- It is a diamond shaped mass of liver cells from two adjacent hepatic lobules.
- It has a central vascular core formed of
  a. terminal branches from portal vein
  b. hepatic artery
  c. a bile ductule.
- At either end of the acinus, is a central vein.
- The hepatic acinus can be subdivided into three zones according to its blood supply:
  - Zone I: comprises cells closest to the blood vessels, consequently this zone is r ichly supplied by blood with excellent content of nutrients and oxygen.
  - Zone II: comprises cells intermediate in location and also in their blood supply.
  - Zone III: comprises the peripherally located cells which have the poorest blood supply almost exhausted of nutrients and oxygen and highly charged with meta bolites.

10. Give an account on the exocrine portion of the pancreas. *(Old exam. Do not panic ☺)*

The exocrine pancreas secretes an alkaline digestive juice containing water, ions, enzymes and proenzymes e.g. trypsinogen, chymotrypsinogen, peptidases, nucleases, lipases and amyl ases

It is composed of acini and ducts

The Pancreatic Acini:

- Serous acini
- Generally larger than those of the salivary glands with a narrow lumen
- Oval in shape. Yet, being overcrowded within the lobule and lying back-to–back against each other, the acini press against one another thus acquiring variable shapes
- Has a characteristic staining pattern, where its central zone appears acidophilic and the periphery is deep basophilic.
- The acini are lined by pyramidal cells with indistinct cell boundaries. Their nuclei are rounded and situated eccentrically nearer to the base.

Ultrastructure: (features of a typical protein synthesizing cell).

- Its basal part is occupied by a large number of rough endoplasmic cisternae which comprise for the peripheral basophilia of the acini.
- Many basal filamentous mitochondria which give the basal region a striated appearance.
- A prominent Golgi complex occupies the supranuclear region.
- The apical cytoplasm is crowded with secretory zymogen granules that cause the central eosinophilic staining of the central zone of the acini.
- The apical cell membrane bears short microvilli
- The lateral membranes exhibit well developed epithelial junctional complexes.
- The lumen of each pancreatic acinus is characteristically lined with flat cells having flat nuclei surrounded by few organelles known as the centro-acinosal cells. They represent the first part of the intralobular ducts (intercalary duct) which becomes telescoped inside the lumen of the acinus.

The Pancreatic Duct System: (A recent exam asked about the Duct System only ☺)

1. The Intralobular Ducts:

- They are very short and therefore are hardly seen in histological sections of the pancreas.
- They are formed of the intercalary ducts, the proximal part of which is telescoped inside the lumen of the pancreatic acinus forming the centroacinosal cells.
- The remaining short part is lined by flat cuboidal cells.

2. The Interlobular and Interlobar Ducts:

- These are seen in the septa between the lobules and lobes and are lined by simple cuboidal and simple columnar cells respectively.

3. The Main Pancreatic Duct:

- It is a larger duct with a wider lumen.
- It is lined by simple columnar cells with goblet cells and occasional enteroendocrine cells in between; a lining which is similar to that of the duodenum where it opens.

*Similar Question from **2013**:*

Mention the histological structure of the pancreatic duct system. (1.5 marks)

11. Tabulate the histological differences between ileum and appendix

12. Enumerate different types of cells lining the fundic glands and mention the function of each.

1- Mucous neck cell
   Mucus isa soluble to lubricate gastric content
2- Oxyntic cell (Parietal)
   HCl formation & secretion
   -production of intrinsic factor to absorp B12
3- Peptic cell (Zymogen)
   Production & secretion of digestive enzymes, pepsin, lipase
4- Entero-endocrine
   Secrete peptide hormone(paracrine,autocrine, endocrine)
5- Undifferentiated (stem cell)
   Upward replacing cells of gastric pits & surface mucous cells
   Downward to new mucous neck cells, parietal,chief & endocrine cells

13. Tabulate the histological differences between the oesophagus and duodenum.

	Oesophagus	***Stomach***
Discription	Muscular organ , 25 cm	Most dilated segment
Place	Greatest part in the thorax except terminal 3-4 cm in abdominal cavity through diaphragm	Add acidic fluid to ingested food, transform it into chyme & digestion by proteolytic enzyme
Function	Transport of masticated food bolus from mouth to stomach	Receives enzymes & alkaline buffer from pancreas and bile from the liver

Mucosa: ***1-Epi*** ***2-Lamina propria*** ***3-Muscularis***	1- Non-keratinized str. Squ.epi. With langerhan's cells to resist friction -continous renewal by mitosis 2-loose CT rich in BV,N & lymphatics. -uppermost part has mucosal oesophgeal glands (tubular, mucous secreting,neutral for protection, lined by columnar cells) 3-S.M -upper third: abscent but represented by elastic fibers & SM bundles -at level of cricoid cartilage: continous layer of longitudinal Sm fibers -lower third: inner circular & outer longitudinal Sm layers	1-it's occupied by intestinal glands (crypts of Lieberkuhn) 2-contain large n. of lymphocytes, macrophages & plasma cells bec. The mucosa is exposed to a lumen containing ingested sub. & bacterial flora 3-underlying the bottom of the Crypt
Submucosa	Dense irrigular C.T has large BV, lymphatics & submucosal oesophgeal glands (compound tubuloalveolar mucous glands, lined by tall culomnar cells, compressed nuclei to the base by mucus droplets, acidic mucous for lubrication)	- Its 1st part is occupied by Brunner's Glands - formed of dense CT. contain Bld capillaries, lymphatics and nerves
Musculosa	- inner circular & outer longitudinal Sm layers -upper third:striated M. / in the middle: mixture/ lower third :Sm only	Conform to their regular structure
Serosa/ Fibrosa	-loose areolar C.T. connects it to surrounding structures (Adventitia) -in abdomen covered by mesothelium	Conform to their regular structure

14. Tabulate the differences between the major 3 salivary glands.

	Parotid	*Submandibular*	*Sublingual*

Capsule & Septa	Thick & well developed	Moderately thick	Thin Well developed
Fat cells	Large number Increase with age	Less numerous	
Secretory Acini	serous	Serous 80%: mucous 20%	99% mucous 1% serous
Intercalary ducts:Secretory striated ducts	50%:50%	20%:80%	1%:99% Very short & poorly developed -the gland possesses multiple ducts that empty into submandibular duct or directly onto the floor of the mouth

15. Compare between the duct system of the parotid gland and the pancreas.

Page 129 & page 132

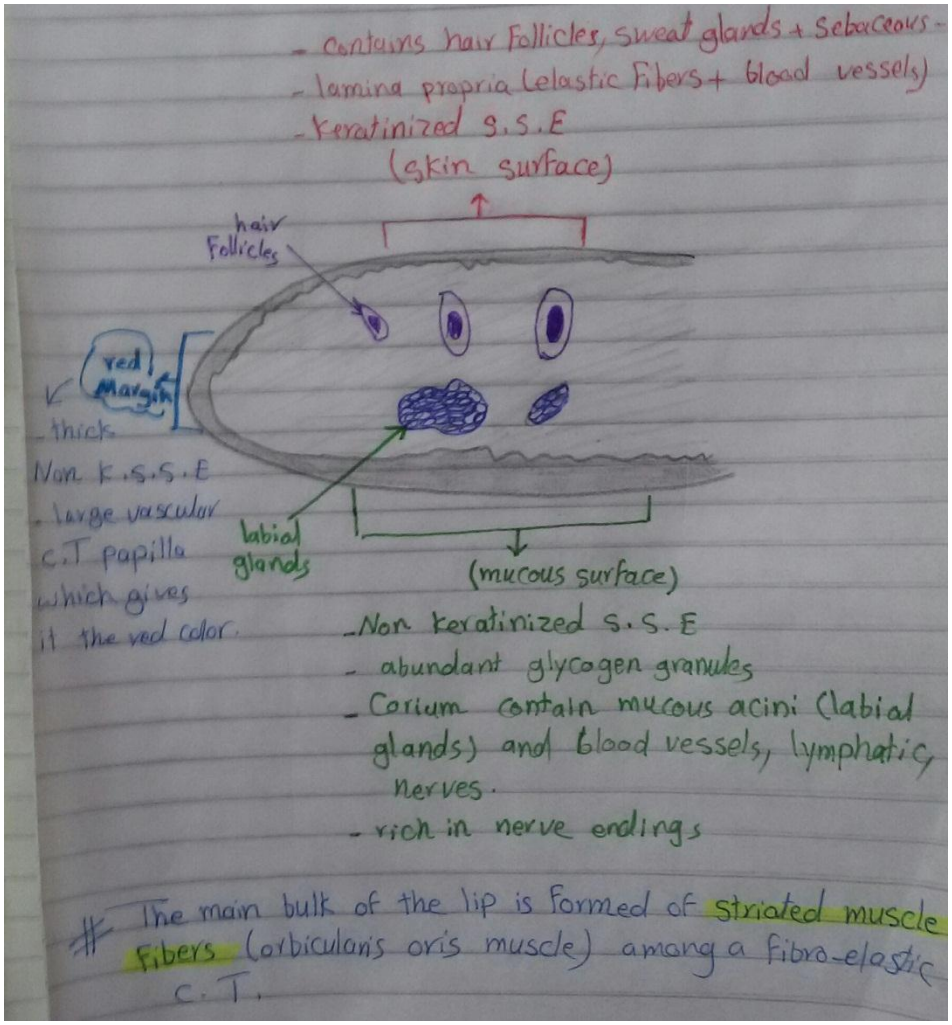
16. Tabulate the points of differences between mucous and serous acini of salivary glands.

	Mucous Acini	*Serous Acini*
Cell number	5-7	3-5

secretion	Mucus (viscid secretion rich in mucin glycoprotein)	Watery secretion rich in salts,proteins & enzymes(amylase,lysozymes ,peroxidase, DND & RNA nucleases)
Glands	Sublingual & Palatine Glands	Parotid & Von Ebner's Glands
Histological section	Pale, large,rounded or oval with relatively wide lumen	Small,deeply stained,rounded with narrow lumen
Epi.Lining	Irrigular,broad cuboidal or pyramidal with distinct boundries	Short pyramidal cells with Indistinct boundries
Nuclei	Flattened basal	eccentric
Cytoplasm	Pale vacuolated due to dissolution of mucinogen granules	
Organelles	At the base:few arrays of rER,few mitocondria,well developed Golgi Apex: croweded mucigen granules	At the base:few arrays of rER,few mitocondria,well developed Golgi,free ribosomes Apex: croweded secretory granules(zymogen)
Basketcells	Numerous to help drainage of the thick mucus into the ducts	Few

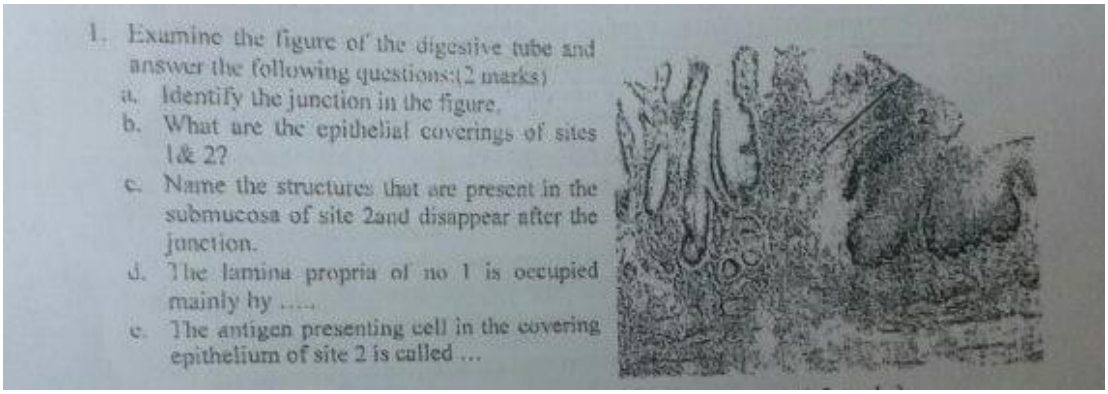
17. Draw a labeled histological diagram of a section in the lip

مش علينا رسم ، بس ممكن لو جه سؤال محتاج منك وصف كتير ترسمله diagram مبسط كده اختصارا للوقت



18. Mention the histological changes at the gastro-esophageal junction.

19.



I- THE GASTRO-OESOPHAGEAL JUNCTION

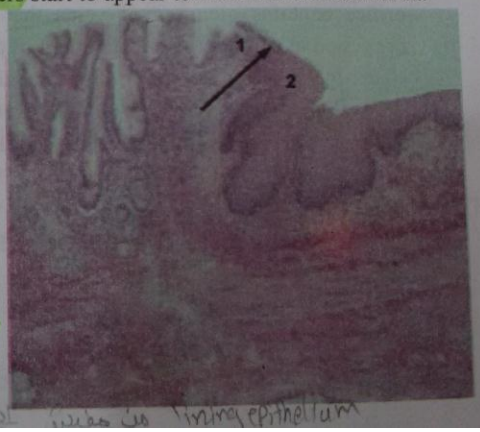
1- The epithelium changes abruptly from the non keratinized stratified squamous type, lining the oesophageal end, to the simple columnar epithelium which is interrupted by gastric pits.

2- The corium is widen and shows simple branched tubular glands of the cardiac end of the stomach.

3- The oesophageal mucous glands stop, or continue for a variable distance into the submucosa of the stomach cardia.

4- Additional oblique smooth muscle fibers start to appear towards the stomach cardia to form the innermost layer of the musculosa.

5- The terminal end of the oesophagus as it passes through the diaphragm and becomes an abdominal organ to join the stomach, it acquires a mesothelial covering. Accordingly, this layer will be formed of loose connective tissue covered by simple squamous epithelium as the serosa of the remaining parts of the digestive tube.



a- Gastro-esophegeal junction
b- 1) simple columnar epithelium
   2) non keratinized S.S.E
c- submucosal esophageal glands
d- simple branched tubular cardiac glands
e- langerhan's cells

3- PARA-GIT

1. Give one word for the following:

- A technique used to detect the larvae of Anklystoma and Strongoloides in the soil.

Baermann's technique

- A disease caused by the larval stage of Taenia solium.

cysticercosis

2. T/F:

- The infective oocyst of Cryptosporidium has a double wall and 8 sporozoites.

False (oocysts contain four naked sporozoites)

3. Give the reason for:

Intestinal amaebiasis can cause dangerous complications.

- As further progress of invasion may occur by amoeba penetrating muscular layer of large intestine & reaching serous coat causing **peritonitis**.
- Amoebiasis of caecum may extend to appendix causing amoebic **appendicitis** .
- In chronic infection with superadded bacterial infection a granulomatous nodular mass develops (**amoeboma**)
- Erosion of blood vesels in the wall of ulcers causes **haemorrhage**
- & amoeba may be carried by blood as emboli to liver (amoebic hepatitis, amoebic liver abscess) & other organs(pulmonary amoebiasis , cutaneous lesions , cerebral lesions.

4. Differentiate between different types of cercariae witn an example for each type.

leptocercous	furcocercous	pleurolophocercous
Body in it organization resemble adult fluke.	Body in it organization resemble adult fluke.	Body in it organization resemble adult fluke.
Tail : simple	Forked at its end	Provided with a fin or membrane
Example: cercaria of fasciola hepatica	Cercaria of schistosoma mansoni	Cercaria of heterophyes heterophyes

5. Mention a parasite causing the following manifestation and state the infective stage and drug of choice:

- **Rectal prolapsed.**

TRICHURIS TRICHURA

Embryonated eggs

Mebendazole

- **Steatorrhea.**

Strongyloide stercoralis

Filariform larva

Ivermectin

- **Nocturnal perianal itching.**

Enterobius vermicularis

Embyonated eggs

Mebendazole

ALSO For symptomatic relief of anal itching, apply 1% phenol or 1% white precipitate
ointment around the anus at bedtime. It helps to kill the emerging worms or eggs
and in the same time prevent secondary infection.

- **Frothy pale diarrhea (lentil soup).**

Giardia lamblia
Cyst of giardia lamblia
metronidazole

6. State the uses of enterotest.

A type of test that may be used to detect presence of parasites in the upper part of small intestine(as a part of laboratory diagnosis techniques) such as:
fasciola hepatica (detect eggs of fasciola)
Giardia lamblia(trophozoites)
Strongyloides stercoralis (rhabditiform larva)
Cryptosporidia(oocysts)

7. Where can you find: coracidium of diphylobothrium latum.

In fresh water in lakes areas.

(It's not present in Egypt)

8. A patient with an acute onset of fever and stabbing right hypochondral pain showed on examination a muddy complexion, an enlarged tender liver and right intercostals edema.

a) What is the possible parasitic cause?

Entamoeba histolytica

b) Mention the methods of diagnosis.

- History & clinical picture of the patient

- Immunologically >> IHAT , ELISA & gel diffusion tests are positive in amoebic abscees(invasive amoebiasis)
- x-ray shows upward displacement of diaphragm with reduced movement & sometimes fluid level can be seen
- ultrasonography
- CT scan
- Aspiration is indicated only if there is a large abscess . aspirated material is viscid , chocolate brown and thick(anchovy-sauce) . trophozoites are never found in this pus except in pus taken from the edge of the abscess
- Trophozoites can be found in sputum if liver abscess ruptures in lung

c) Mention the infective stage.

Mature four nucleated cysts

d) Mention the drug of choice.

Specific treatment is **metronidazole.**

*hospitalization & bed rest are essential.

*when size o abscess is large, needle aspiration is advisable.

9. Mention the parasite with the following characteristics in their life cycle. Describe the infective stage.

- **Two important nematodes with larval migration in the lung**.

❶ Ascaris lumbricoides
- ⇨ Mature egg containing 2nd stage rhabditiform larva

❷ Strogyloides Stercolaris
- ⇨ 3rd stage filariform larva

❸ Anchylostoma Duodenale
- ⇨ 3rd stage filariform larva

منننساش نقول الشكل أو نرسمه ..

- **A trematode maturing in the portal tract.**

❶ Schistosoma mansoni

⇨ Furocercous cercaria

10. Mention the role of the following in causing or transmitting disease to man:

- Pigs.

① Raw pork meat => cysticercus cellulosae => Taenia solium
(pigs are intermediates hosts)

② Contact with pigs => cyst (contamination of food and drinks) => Balantidium coli
(It's basically a parasites of pigs)

11. Give two important differences between:

- Life cycle of Ascaris lumborcoides and enterobius vermicularis.

POC	Entrobius vermecularis	Ascaris lumborcoides
Infective stage	Larvated egg	Larvated egg
Diagnostic stage		Immature egg
Definitive host	Man	Man
Intermediate host	Not present	Not present "soil transmitted"

Hatching	Intestine	Intestine
Mode of infection	• Hands • Inhalation • Polluted food • External autoinfection	• Ingestion of infected food or drinks • Hands and house flies

- True and false fascioliasis.

POC	True	False
Mode of infection	Eating infected foods or drinks	Eating infected livers
Infective stage	Encysted metacercaria	Stages other than encysted metacercaria
Clinical pic.	Typical clinical pic.	No symptoms
Prognosis	Heals after treatment	No disease No treatment Just avoid eating livers
After 3 days	Immature eggs in the stool	Free stool

12. Mention the medical importance of Pirenella conica and Lymnea snail.

Pirenella conica => (intermediate host) => Heterophyes heterophyes
تدخل
mature egg
تطلع
pleurolophocercus Cercaria

Lymnea snail spp. => (intermediate host) => Fasciola hepatica (trunculata) and gigantica (natalensis)
تدخل
meracidium
يطلع
leptocercus Cercaria

13. Give reasons:

- Cyst of Entamoeba histolytica is not found in the stools of patients with acute amoebic dysentery.
Acute amoebic dysenteric phase is characterized by frequent defecation
4-8 times/day so the trophozoites don't have time to allow encystation.

- Egg of Enterobius is rarely found in stool.

- Isospora belli is dangerous in immunocompromised patients.
In immunocompromised patients there is no cellular immunity so no infiltration of lymphocytes, eosinophils and plasma cells at the site of inflammation and distortion of intestinal villi.
So causes profuse diarrhea, weakness, anorexia and weight.

- Appendicitis may occur in Ascaris lumborcoides infection.
Because of adult worms wandering habits.
A single worm may reach abnormal foci like appendix and causes acute symptoms (appendicitis).

14. Mention two uses for mebendazole.

① Trichuris trichura
② Ascaris lumbercoides
③ Entrobius vermecularis

15. Differentiate between:

- The diagnostic stages of Cryptosporidium and Isospora in fresh fecal samples.
Cryptosporidium => Thick walled oocyst
Isospora => Immature oocyst

- Anemia caused by Diphylobothrium latum and Anklystoma duodenale.
Diphylobothrium latum => pernicious anemia
Anklystoma duodenale => Microcytic hypochromic

- Capillaria hepatica and taenia solium as regards habitat, infective stage and mode of infection.

POC	Taenia saginata	Taenia solium
Segments morphology	- 15-20 lateral branches - 1000-2000 segments - 300-400 testes	**- 7-13 lateral branches** **- 1000 segments** **- 150 testes**

- Two cestode that can be detected in human stools as regards diagnostic morphology and intermediate host.

POC	Capillaria phillipensis	Taenia solium
Habitat	Small intestine "jejunum" mainly (man + fish eating birds)	Small intestine (man)
Infective stage	Larva	Cysticercus cellulosae
Mode of infection	Improperly cooked infected fish	Improperly cooked infected pork meat

	- bilobed ovary	**- trilobed ovary**
Intermediate host	Cattle	**Pigs or humans**

16. Mention the parasitic stages responsible for:

- Loffler's syndrome.
Larval stage of Ascaris lumbercoides

- Swimmer's itch.
Cercaria of Schistosoma mansoni
/: الدكتور قالت الاسم ده في المحاضرة

- Muscle pain 3 weeks after eating pork meat.
Cystocercous cellulosae
D:المفروض ده باثو بيولوجي
مش علينا الـ
muscle pain
.. بتاعها في الموديول ده

17. Describe the stages found in the stool samples taken from:

- Diarrhea caused by Giardia lamblia.
Trophozoites
.. + نقول شكله إيبه أو نرسمه

- Dysentery caused by Balantidium coli.
Trophozoites + or – cysts
.. بردو نقول الشكل أو نرسمه

- Stronglyloides stercoralis infection
* Rhabditiform larva => normal bowel

* 3rd stage filariform => constipation
Non sheathed
Notched tail
Esophagus = 1/2 the length

* Partially embryonated eggs => diarrhea
Thin shelled

Give a reason :

Bilharizial corpulmonale occurs in schistosomiasis mansoni.

It occurs late as a complication when the portal pressure rises more than the systemic pressure. So blood will pass from the portal circulation to the systemic carrying *Schistosoma mansoni* ova to reach the lungs.

Contact with food handlers paing Taenia eggs in his stool is not dangerous.

As Taeniasis in man occurs by **ingesting infected meat containing cysticercus bovis** which is the **infective stage not the eggs .**

portal hypertension as a complication of S.mansoni infection.

As a result of hepatic affection which is in the form of bilharizial hepatic fibrosis

Meat should be properly cooked before eating.

As an individual prophylaxis to prevent occurance of taeniasis which occurs due to consumption of imperfectly cooked containing the infective stage which is :

Cysticercus bovis (beef meat)

Cysticercus celluloae(in pig's meat)

Myocarditis may occur in Heterophyes heterophyes infection

As eggs may sometimes migrate by lymphatics to the heart causing myocarditis & heart failure

Praziquantel can be used in treatment of hymenolepis nana

Since it is systematically absorbed thus acting on
both the cysticercoid in the villi and adults in the lumen of the intestine.

Intestinal obstruction may occur in ascaris infection

- As ascaris lumbricoides worms are large nematodes (males 15-30 cm , females 20-40cm)
- and have the tendency to **wandering habits** especially when irritated with spicy foods, fever, drugs such as anesthetics.
- Aggregate masses of worms may cause volvulus or intestinal obstruction
- Wandering single worms may reach abnormal foci and cause acute symptoms e.g. obstruction of bile duct, pancreatic duct, appendix, liver abscesses, intestinal perforation etc.

odema occurs in capillaria phulippinensis infection

because Worms invade the small intestine causing severe entropathy, which is manifested
by derangement of intestinal function with malabsorption and **loss of fluid**, **protein** and
electrolytes in the intestinal tract.

Mictocytic hypochromic anemia occurs in ancylostoma duodenale infection.

- As when young worm reaches the small intestine,it attaches itself to the mucosa with its strong buccal capsule and teeth digesting part of the villi which they have sucked in their mouth.
- Blood is sucked out of the tissues but a greater amount is lost at the site of attachment due to the anticoagulant secreted by the worms.
- estimates of blood loss per worm are 0.15 ml/day. Up to 200ml of blood may be lost by the patient with heavy infection, but around 40% or so of the iron may be reabsorbed before it leaves the intestine.
- In addition to malabsorption & malnutrition.

Caecum is the commonest site of Ehistolytica infection
As the most affected areas in the colon are the parts in which there's sluggish movement of faecal matter like caecum . in addition to Rt. & Lt. colic flexures & sigmoid colon.

Cryptosporidia differs from other coccidian protozoa

- Organisms are present just beneath brush border of epithelial cells of gastric & small intestine mucosa (intracellular – extracytoplasmic)
- Oocysts contain four naked sporozoites without sporocysts (immediately infective when excreted)
- Internal autoinfection may occur

Mention the parasites which can be diagnosed by the following techniques .

AND Mention parasitic stage that can be found in each.

Rectal swab : Schistosoma mansoni >> mature eggs

anal swab : enterobius vermicularis >> mature eggs

Entero test capsule :

fasciola hepatica (detect eggs of fasciola)
Giardia lamblia(trophozoites)
Strongyloides stercoralis (rhabditiform larva)
Cryptosporidia(oocysts)

Name the parasite(s) tha can cause the following

Appendicitis :

Taenia saginata / trichuris trichura / ascaris lumbricoides /
E. histolytica.

Halzoun > adult living worms of fasciola hepatica

Rectal 42rolapsed > trichuris trichura

Flask-shaped ulcer in small intestine > Entamoeba histolytica / balantidium coli

Enumerate 2 parasites causing the following :

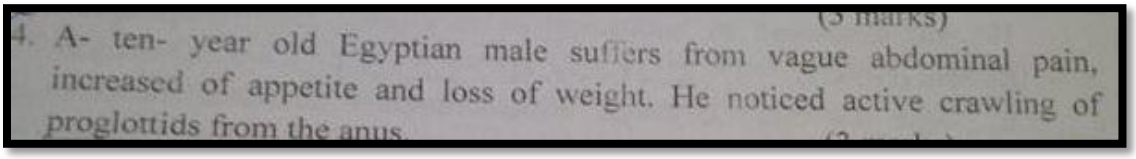
And tabulate the differences between them as regard infective & diagnostic stage.

a)steotorrhea

Strongyloides stercoralis	Giardia lamblia
Infective s. >> 3rd stage filariform larve	cysts
Diagnostic s. >> rhabditiform larva in stool	In diarrhoeic stool >> trophozoites In well-formed stool >> cysts

b)anaemia

Trichuris trichura	Anchylostoma duodenale
Infective s. > embryonated eggs	3rd stage filariform larva
Diagnostic s. >unembyonated egg in faeces	Eggs in faeces


4. A- ten- year old Egyptian male suffers from vague abdominal pain, increased of appetite and loss of weight. He noticed active crawling of proglottids from the anus.

Questions:

Mention parasite responsible for this case.

Taenia saginata

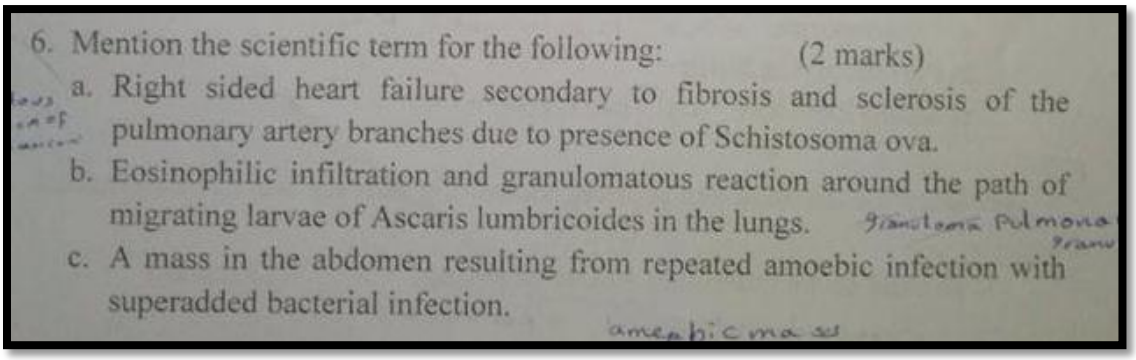
How did he acquire infection?

Ingestion of imperfectly cooked beef meat containing cysticercus bovis (infective stage)

What are possible complications of the case?

Intestinal obstruction

Acute or subacute appendicitis or cholangitis



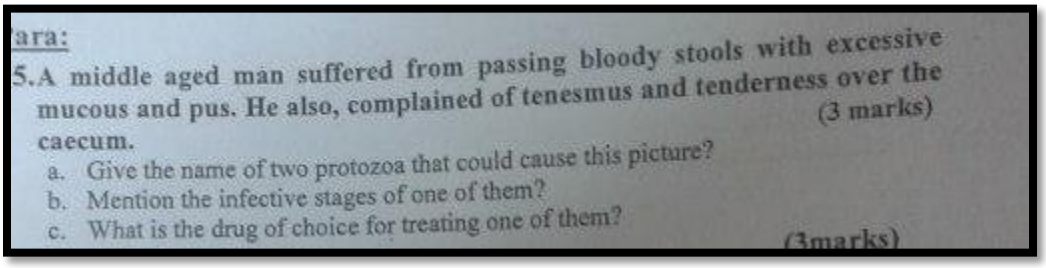
6. Mention the scientific term for the following: (2 marks)
   a. Right sided heart failure secondary to fibrosis and sclerosis of the pulmonary artery branches due to presence of Schistosoma ova.
   b. Eosinophilic infiltration and granulomatous reaction around the path of migrating larvae of Ascaris lumbricoides in the lungs.
   c. A mass in the abdomen resulting from repeated amoebic infection with superadded bacterial infection.

Answers:

a-bilharizial corpulmonale

b-ascaris pneumonitis

c-amoeboma



ara:
5. A middle aged man suffered from passing bloody stools with excessive mucous and pus. He also, complained of tenesmus and tenderness over the caecum. (3 marks)
   a. Give the name of two protozoa that could cause this picture?
   b. Mention the infective stages of one of them?
   c. What is the drug of choice for treating one of them? (3marks)

a-Entamoeba histolytica	a-balantidium coli
b-mature four nucleated cysts	b-cyst of b.coli

c-drug of choice for treating infection with balantidium coli is >>> tetracycline

In a table differentiate between one protozoa & one helminth causing malaborption as regards **infective stage** & **mode of infection**

Helminth (strongyloides stercoralis)	Protozoa (Giardia lamblia)
Infective Filariform larva	cysts
-Penetration of intact skin by infective stage. -**Autoinfection** can sometimes	-infection occur through swallowing the cystic stage by: • Contamination of food or drink

occur in strongyloidiasis especially if the patient is constipated. • **Internal autoinfection** happens when some of the rhabditiform larvae moult to the infective filariform larvae while passing down the bowel and reinfect the host by invading the intestinal mucosa to reach the lungs via the portal system. • **External autoinfection** occurs when rhabditiform larvae in fresh stools transform to the infective filariform larvae during their passage through the anal canal and these penetrate the perianal or perineal skin. • When the parasite when the patient becomes **immunosuppressed** then **hyperinfection** results and the infection flares up with large numbers of larvae in all tissues of the body.	• Direct infection from man to man • Autoinfection • Mechanical transmission by house flies

4- Anatomy (GIT)

1- Give the embryological origin of

Meckel's diverticulum:

(Meckl's diverticulum: It is due to persistence of small pouch of the yolk stalk. Length: 2 inches. Distance from caecum: 2 feet .Incidence: 2% of people)

Since it is one of the congenital anomalies of small intestine its origin will be:

- Endoderm of midgut : mucosa & glands
- Splanchnic secondary mesoderm : submucosa & musculosa and serosa

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2- Discuss the development and anomalies of stomach.

Origin:

Endoderm of foregut: mucosa & its glands.

Splanchnic secondary mesoderm: submucosa, musculosa and serosa.

Development:

Fusiform part of foregut:

1- Its dorsal border grows more → greater curvature.
2- Its dorsal border grows less → lesser curvature.
3- Most cranial part of dorsal border grows rapidly → fundus.
4- Rotation of stomach 90 degrees clockwise around its longitudinal axis →Left surface becomes anterior & right surface becomes posterior. So left vagus becomes anterior gastric nerve & right vagus becomes posterior gastric nerve.
5- The stomach was first vertical→by development of liver → its long axis will be oblique.

Congenital anomalies:

1- Thoracic stomach: associated with short esophagus.
2- Congenital pyloric stenosis: due to hypertrophy of the circular muscles in the pyloric region → projectile vomiting of the infant after feeding.
3- Hourglass stomach.
4- Transposition of stomach (may be associated with situs inversus)

3- Mention the relations and arterial supply of pancreas.

Relations:

a) Head of pancreas:

- It lies in the concavity of the duodenum:
  Superiorly it is related to the 1st part of duodenum, 2nd part on the right side (separated from it by superior & inferior pancreatico-duodenal arteries), and 3rd part inferiorly.
- Anteriorly: it is related to transverse colon.
- Posteriorly: it is related to IVC, renal veins and common bile duct.
- Uncinate process lies between abdominal aorta and superior mesenteric vessels.

b) Neck of pancreas:

- Anteriorly: it is related to gastro-duodenal junction.
- Posteriorly: it is related to the formation of portal vein from splenic and superior mesenteric veins.

c) Body (triangular in cross section):

- It has three surfaces (anterior, posterior and inferior) and three borders (anterior, superior and inferior).
- **Surfaces:**

i. **Anterior surface:** Related to stomach, separated from it by the lesser sac.
ii. **Inferior surface:** Related to duodeno-jejunal flexure, loops of ileum and end of transverse colon (from right to left).
iii. **Posterior surface:** It is related to posterior abdominal wall:

a. Aorta and origin of sup. Mesenteric artery
b. Splenic and left renal vein
c. Left psoas major
d. Left crus of diaphragm
e. Left kidney
f. Left supra renal gland
g. Left sympathetic chain

- Borders:

1. **Superior border:** It is related to splenic artery.
2. **Anterior border:** It gives attachment to transverse mesocolon and greater omentum.
3. **Inferior border:** It separates the inferior from the posterior surfaces.

d) Tail of pancreas:

- It is related to the visceral surface of spleen near its hilum.
- It reaches the hilum via the lieno-renal ligament.

Blood supply:

- Arterial supply:

1. Superior, inferior pancreatico-duodenal arteries: to the head.
2. Pancreatic branches of splenic artery: to the rest of pancreas.

- Venous drainage: To splenic vein and portal vein**.**

4- **Discuss:**

a) Origin, course and branches of the superior mesenteric artery.

1. It arises from the front of the aorta 1/2 inch below the celiac trunk at the level of the lower border of the body of first lumbar vertebra.
2. It is directed downwards behind the neck of the pancreas. With the superior mesenteric vein on its right side then it lies in the groove between the neck and the uncinate process of the pancreas.
3. The two vessels then pass over the third part of the duodenum and enter the upper end of the mesentery of the small intestine.
4. They pass down to the right along the root of the mesentery and end at the ileum 2 feet proximal to the caecum.

Branches:

1. **Inferior pancreatico-duodenal artery:**

   It supplies the duodenum below the entrance of the bile duct. It runs in the curve between the duodenum and the head of the pancreas and anastomoses with the superior pancreatico-duodenal artery.

2. **Jejunal arteries:**

   They arise from the left of the main trunk and pass forward between the two layers of the mesentery. They join each other in a series of anastomosing loops which form single arterial arcades in the upper part of the jejunum, double arcades down. From the arcades straight arteries pass to the mesenteric border of the jejunum.

3. **Ileal arteries:**

   They enter the mesentery and form a series of arterial arcades. Straight vessels (vasa recta) pass to the mesenteric border of the ileum. The arcades of the terminal ileal branch anastomose with those of the terminal part of the main trunk of the superior mesenteric artery

4. **Ileo-colic artery:** It arises from the right side of the superior mesenteric trunk low down in the base of the mesentery. It runs to the ielo-colic junction, where it gives off:

   a) Ileal branch: It anastomoses with the terminal branch of the superior mesenteric artery.

   b) Colic branch: It runs up along the left side of the ascending colon to anastomose with the right colic artery.

c) Anterior coecal artery: It ramifies over the anterior surface of the caecum.
d) Posterior coecal artery: It supplies the posterior wall of the caecum.
e) Appendicular artery: It passes towards the tip of the appendix in the mesoappendix.

5. **Middle colic artery** passes forwards between the two leaves of the transverse mesocolon and at the intestinal border of the transverse mesocolon it divides into right and left branches which run along the transverse colon. The right branch anastomoses with the ascending branch of the right colic artery. The left branch anastomoses with a branch of the left colic artery.

6. **Right colic artery:** It arises in the root of the mesentery from the right side of the superior mesenteric artery. It runs to the right and divides near the left side of the ascending colon into two branches.
   a) The descending branch runs down to anastomose with the colic branch of the ileo-colic artery.
   b) The ascending branch runs up to anastomose with a branch of the middle colic artery.

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b) Boundaries and contents of the inguinal canal.

It is an oblique inter-muscular slit 6 cm long above the medial half of inguinal ligament.

- It begins at the deep inguinal ring and terminates at the superficial ring.
- Anterior wall:

Aponeurosis of the external oblique muscle along the whole length of the canal and fleshy fibers of internal oblique muscle along the lateral 1/2 of the canal.

- Posterior wall:

It is formed by transversalis fascia along the whole length of the canal, conjoint tendon along medial 1/2 and reflected ligament along medial 1/4.

- Superior wall (roof):

Arching fibers of the internal oblique and transverse muscles.

- Inferior wall (floor):

Inguinal and lacunar ligaments.

- **Contents:** it transmits the spermatic cord or the round ligament of the uterus and the genital branch of the genitofemoral nerve, both of which also run through the deep inguinal ring and the inguinal canal. An indirect inguinal hernia (if present) also passes through this canal.

c) **Attachments, relations and nerve supply of psoas major.**

Psoas Major

- **Origin**: Transverse processes of lumbar vertebra; lateral surface of bodies of T12-L5 and intervening IV discs.
- **Insertion:** By a strong tendon to lesser trochanter of femur.
- **Innervation**: Lumbar plexus via anterior rami of L1-2-3 nerves.
- **Actions**:
  - Flexion of the thigh.
  - Lateral flexion of the trunk.

5- Mention the surface anatomy of the gall bladder fundus

Fundus is at the tip of the right ninth costal cartilage

6-Name two main vessels lying posterior to the 1st part duodenum.

Portal vein & gastro-duodenal artery

7-Which part of the peritoneal sac separates the stomach from its bed?

Lesser sac

Name the main big vessel in the stomach bed.

Splenic artery

8-Where does the common bile duct open?

It joins the pancreatic duct to form the ampulla of vater which opens in the posterior wall of the 2nd part of the duodenum below its middle .The opening is guarded by the sphincter of Oddi.

9-Name the muscles forming the lateral wall of the ischiorectal fossa.

Obturator internus muscle, obturator fascia & pudendal canal.

10-Name and give the origin of arteries supplying the duodenum.

Arterial supply of the duodenum:

1. Supra-duodenal artery: from the hepatic artery proper (celiac trunk).
2. Superior pancreatico-duodenal artery: from gastro-duodenal (celiac).
3. Inferior pancreatico-duodenal artery: from superior mesenteric artery.

11- Name the branches of the inferior mesenteric artery and tributaries of the portal vein.

Branches of inferior mesenteric artery:

a) Left colic artery: it divides into 2 branches the upper branch which passes to the splenic flexure & lower branch passes transversely to the descending colon.

(Each of the arteries divides into ascending and descending branches which anastomose with middle colic artery and with each other)

b) Sigmoid arteries

c) It continues as superior rectal artery

Tributaries of portal vein:

a) Superior mesenteric vein
b) Splenic vein (receives from inferior mesenteric vein)
c) Right & left gastric veins
d) Paraumbilical vein
e) Cystic vein

12- Give the origin and insertion of the quadrates lumborum muscle.

Origin: Iliolumbar ligament, internal lip of iliac crest and transverse processes of L5.
Insertion: Medial half of inferior border of 12th rib and tips of lumbar transverse processes.

13- Name the nerves related to the medial border of psoas major.

The obturator nerve

The nerve arises from anterior division of the L2, 3, 4 anterior primary rami. It emerges medial to the psoas muscle and leaves the pelvis through the obturator foramen.

The lumbosacral trunk

A part of the anterior division of the L4 primary ramus and the L5 primary ramus form the lumbosacral trunk. The trunk lies anterior to the ala of the sacrum to join the S1 anterior primary ramus

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14- Give the vertebral level of:

- Aorta bifurcation:

Forth lumbar vertebra (L4)

- Pylorus of the stomach:

1/2 inch to the right of the median plane at level of L1 (transpyloric plane)

- Beginning of IVC:

To the right of 5th lumbar vertebra (L5)

- Superior mesenteric artery:

From the front of aorta 1/2 inch below the celiac trunk at the level of the lower border of the body of the first lumber vertebra (L1)

15- Name the veins establishing a porto-systemic anastomosis half way down the anal canal.

- Superior rectal (portal)
- Middle & inferior rectal (systemic)

16- Describe the blood supply of the large intestine.

a) Caecum :
   - Arterial supply : anterior and posterior **caecal branches** from ileocolic artery (from superior mesenteric artery)
   - Venous drainage : into superior mesenteric vein into portal vein

b) Appendix:
   - Arterial supply : **Appendicular artery** from posterior caecal artery from ileocolic artery
   - Venous drainage : superior mesenteric vein

c) Ascending colon :

- Arterial supply : **ileocolic & right colic** arteries from superior mesenteric
- Venous drainage: veins corresponding to arterial supply.

d) Transverse colon :
- Arterial supply :
  Right 2/3**: right & middle colic** arteries of superior mesenteric
  Left 1/3: **ascending branch of left colic** artery from inferior mesenteric
- Venous drainage: veins corresponding to arterial supply.

e) Descending colon :
- Arterial supply :**upper and lower left colic** arteries from inferior mesenteric artery

f) Sigmoid colon :
- Arterial supply : **sigmoid branches** from inferior mesenteric artery
- Venous drainage: veins corresponding to arterial supply.

g) Rectum :
- Arterial supply :

1. **Superior rectal artery**: It is the continuation of **inferior mesenteric artery.** It supplies the rectum and upper half of anal canal.
2. **Middle rectal artery**: It arises from the anterior division of internal iliac artery.
3. **Inferior rectal** artery: It arises from internal pudendal artery.

- Venous drainage**:**

1. **Superior rectal vein** continues up as inferior mesenteric vein which drains into the splenic vein. (Portal circulation)
2. **Middle rectal vein:** Drains into internal iliac vein.(Systemic circulation)
3. **Inferior rectal vein:** Drains into internal pudendal vein. (Systemic circulation)

h) Anal canal:

	Upper part	Lower part
Blood supply	– It is supplied by superior rectal artery. – It is drained by superior rectal vein **(portal circulation)**.	– It is supplied by: 1. Middle rectal artery of internal iliac artery. 2. Inferior rectal artery of internal pudendal artery. – The corresponding veins drain into internal iliac vein **(systemic circulation.)**

17-Give an account on:

- Epiploic foramen.

It is the opening of the lesser sac to the greater sac which lies behind the free edge of the lesser omentum.

- **Boundaries of the epiploic foramen:**

✓ Posterior: the inferior vena cava lies immediately behind the posterior peritoneum.
✓ Superior: the inferior surface of the liver, the caudate process.
✓ Inferior: the first part of the duodenum.
✓ Anterior: the free edge of the lesser omentum; in which the portal vein lies behind, the common bile duct in front and the hepatic artery in front on the left of the bile duct.

- Attachments and contents of the mesentery of small intestine.

- **Attached border** (root of mesentery): It is 15 cm long, extends from duodeno-jejunal flexure to iliocecal junction.

Structures crossed by the root of the mesentery (7):

1. 3rd part of duodenum
2. Abdominal aorta.
3. IVC.
4. Right psoas major.
5. Right ureter.
6. Right gonadal vessels.
7. Right genitor-femoral nerve

- **Contents of the mesentery: (7)**

1- Superior mesenteric vessels in its root.
2- Jejunum and ileum in the free border.
3- Jejunal and ileal areteries and their arterial arcades.
4- Extraperitoneal tissue and fat.
5- Sympathetic nerve fibers.
6- Lymph vessels (lacteals).

- Lumbar plexus.

- Formation: formed by anterior rami of L1-L3, a part of anterior rami of T12 and upper part of L4.

- Position: lies within substance of psoas major. Therefore, relative to the psoas major muscle the various branches emerge either:

– Anterior: genitofemoral nerve
– Medial: obturator nerve
– Lateral: Iliohypogastric, Ilioinguinal, femoral nerve and lateral cutaneous nerve of the thigh.

(Add small details about each nerve ;))

The Iliohypogastric nerve
The Iliohypogastric nerve is formed by fibers from L1, with some contribution from T12. It supplies the skin over the lateral gluteal region and the skin above the pubis.

The Ilioinguinal nerve
The Ilioinguinal nerve is formed in common with the Iliohypogastric nerve. It passes through the superficial inguinal ring.

The genitofemoral nerve
The genitofemoral nerve is formed from L1,2 and passes through the psoas to emerge on its anterior surface. It runs downwards on the psoas and divides into genital and femoral branches.

Lateral cutaneous nerve of the thigh
The lateral cutaneous nerve of the thigh emerges at the lateral border of the psoas muscle. It is formed from the posterior division of the L2, 3 anterior primary rami. It supplies the skin on the lateral part of the thigh.

The femoral nerve
The femoral nerve arises from posterior division of the L2, 3, 4 anterior primary rami.

The obturator nerve
The nerve arises from anterior division of the L2, 3, 4 anterior primary rami. It emerges medial to the psoas muscle and leaves the pelvis through the obturator foramen.

The lumbosacral trunk
A part of the anterior division of the L4 primary ramus and the L5 primary ramus form the lumbosacral trunk.

Muscular nerves
The T12 and lumbar primary rami send short nerves into neighboring muscles; the quadratus lumborum, and psoas.

- Sensory supply of anterior abdominal wall above the umbilicus.

- The skin and muscles of the anterior abdominal wall are supplied mainly by the ventral rami of the **inferior six thoracic nerves** (i.e., the continuation of the **lower intercostals nerves**, T7 to T11) and the **Subcostal nerve** (T12).
- The inferior part of the abdominal wall is supplied by two branches of the ventral ramus of the first lumbar nerve via the **Iliohypogastric** and **Ilioinguinal nerves**.

- Components of the conjoint tendon.

It is the fused lower part of internal oblique and transversus abdominis.
Known as: falx inguinalis. It is the most important part of the posterior wall of inguinal canal.

- Origin of the arteries of the lesser curvature of the stomach.

- **Left gastric artery** arises from the celiac artery.
- **Right gastric artery** arises from the hepatic artery.
- **Short gastric arteries** arise from the splenic artery.
- **Left gastroepiploic artery** arises from the splenic artery.
- **Right gastroepiploic artery** arises from the gastroduodenal branch of the hepatic artery.

18- Mention:

- The anterior relations of the epiploic foramen.

- **Anterior:** the free edge of the lesser omentum; in which the portal vein lies behind, the common bile duct in front and the hepatic artery in front on the left of the bile duct.

- Characteristic features of the large intestine.

The Main Differences between Small and Large intestine:

- **Tenia Coli** are the longitudinal outer muscle layer which is represented by 3 bands. They are not found in the appendix and rectum.
- **Sacculations:** this due the length of tenia coli is shorter than the length of the large intestine.
- **Appendices Epiploicae:** They are small sacs of peritoneum-covered fat hanging from the surface of the colon.

- Structures forming posterior wall of inguinal canal.

- It is formed by transversalis fascia along the whole length of the canal, conjoint tendon along medial 1/2 and reflected ligament along medial 1/4.

19- Enumerate 6 congenital anomalies of the intestine

- Congenital umbilical hernia
- Exomphalos
- Meckl's diverticulum
- Congenital umbilical fistula
- Rotation clockwise → transposition
- Intestinal atresia
- Intestinal stenosis
- Duplication

20- Discuss nerve supply of parotid gland

- **Parasympathetic** secretomotor fibers from the inferior salivary nucleus of the ninth cranial nerve supply the parotid gland. The nerve fibers pass to the otic ganglion via the lesser petrosal nerve. Postganglionic parasympathetic fibers reach the parotid gland via auriculotemporal nerve.
- Postganglionic **Sympathetic** fibers reach the gland as a plexus of nerves around the external carotid artery.

21- Mention surfaces of body of pancreas and relation of each.

Body (triangular in cross section):

- It has three surfaces (anterior, posterior and inferior) and three borders (anterior, superior and inferior).
- **Surfaces:**
  - **Anterior surface:** Related to stomach, separated from it by the lesser sac.
  - **Inferior surface:** Related to duodeno-jejunal flexure, loops of ileum and end of transverse colon (from right to left).
  - **Posterior surface:** It is related to posterior abdominal wall:
    - ✓ Aorta and origin of sup. Mesenteric artery
    - ✓ Splenic and left renal vein
    - ✓ Left psoas major
    - ✓ Left crus of diaphragm

- ✓ Left kidney
- ✓ Left supra renal gland
- ✓ Left sympathetic chain

22-Give a short account on otic ganglion regarding its position, roots and distribution.

- It is a parasympathetic ganglion in the infratemporal fossa below foramen ovale medial to the main trunk of mandibular nerve.
- **Roots:**
  1. **Sensory root:** from mandibular nerve
  2. **Motor root:** from the nerve to medial pterygiod muscle
  3. **Sympathetic root:** from plexus around middle meningeal artery
  4. **Parasympathetic root:** lesser superficial petrosal nerve (from **glossopharyngeal** nerve) (Only the parasympathetic root relays in ganglion)

- **Branches:**
  1. Parasympathetic postganglionic branches to parotid gland
  2. Sensory branches to parotid gland
  3. Sympathetic branches to blood vessels of parotid gland
  4. Motor twig to tensor palati and to tensor tympani

23- Give a short account on the relations and arterial supply of stomach.

- **Relation of Anterior surface: (LAD)**

1- Left lobe of the liver
2- Anterior abdominal wall
3- Diaphragm

- **Relation of Posterior surface (stomach bed):**

It is related posteriorly to lesser sac, diaphragm, spleen, left suprarenal gland, upper part of the left kidney, splenic artery, pancreas, transverse mesocolon and transverse colon

- **Arterial supply :**

- Left gastric artery arises from the celiac artery.
- Right gastric artery arises from the hepatic artery.
- Short gastric arteries arise from the splenic artery.
- Left gastroepiploic artery arises from the splenic artery.
- Right gastroepiploic artery arises from the gastroduodenal branch of the hepatic artery.

24- Discuss parotid gland regarding its relations and lymphatic drainage

- **Lymph drainage:**

The lymph vessels drain into parotid lymph nodes and the deep cervical lymph nodes.

- **Relations :**

- **The superficial (lateral) relations** are parotid lymph nodes, superficial fascia, great auricular nerve and skin.

- **The superior relations** are the external auditory meatus and the posterior surface of tempromandibular joint. The glenoid lobe is directly related to the auriculotemporal nerve.

- **The posteromedial relations** are mastoid process, sternocleidomastoid, posterior belly of the digastric, styloid process and its attached muscles, carotid sheath with internal carotid artery, internal jugular vein and vagus nerve, glossopharyngeal, accessory, hypoglossal, and facial nerves.

- **The anteromedial relations** are the posterior border of the ramus of the mandible, the temporomandibular joint, masseter, medial pterygoid muscle

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25- Give the embryological origin of anal canal and two of its congenital anomalies.

Embryological origin:

- **Upper half :**
  Endoderm of hind gut → mucosa & glands
  Splanchnic secondary mesoderm → submucosa & musculosa
- **Lower half :**
  Ectoderm → stratified columnar epithelium
  Splanchnic secondary mesoderm → submucosa, musculosa & anal sphincters.

Congenital anomalies: imperforate anus – anal stenosis – anal agenesis

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26- Beginning, termination and tributaries of inferior vena cava.

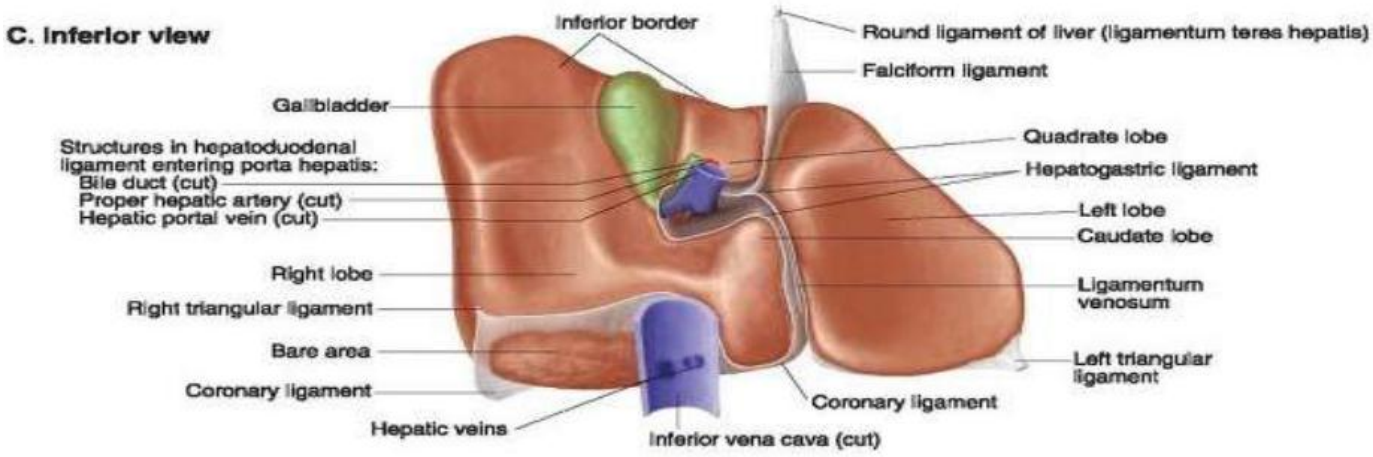
- It is the largest vein in the body. It is formed by union of two common iliac veins anterior to and just to the right of 5th lumber vertebra.
- It ascends on the right side of aorta, passes in the vena cava opening of diaphragm opposite T8 and drains into the right atrium.
  - **Tributaries of I.V.C:**
    1. Two common iliac veins: - they unite together forming I.V.C.
    2. Two pairs of lumbar veins: - 3rd, 4th.
    3. Two renal veins (Rt. & Lt.).

4. Two inferior phrenic veins.
5. Two hepatic veins.
6. Right gonadal vein.
7. Right suprarenal vein.
8. Azygos vein.

27- Draw a diagram showing the relations of the inferior surface of the liver.

Inferior (Visceral) surface: it shows the following features and impressions:

a. Gastric impression
b. Fissure for ligamentum teres
c. Quadrate lobe
d. Fossa for gall bladder
e. Duodenal impression
f. Renal impression
g. Supra renal impression
h. Colic impression
i. Tuber omental (elevated area in the left lobe overlying the lesser omentum).



5- Microbiology

1. Give an account on:

- Diseases caused by cocksackie B virus.

1-Epidemic myalgia (pleurodynia or Bornholm disease)= devil's grip

Symptoms:

1)Acute illness in which patients have fever.

2)A sudden onset of agonizing stitch like pains in the muscles of chest and epigastrium

3)Abdominal pain.

4)vomiting

Prognosis :

The illness is self-limited and recovery is complete though relapses are Common

2-Myocardial and pericardial infections caused by coxsackie B virus

Age :older children and adults but most threatening in newborn

Coxackie B are the most commonly identified agents of viral heart disease in humans.

3-Seroepidemiologic evidence links type I diabetes mellitus (insulin dependent) with coxsackie B .

4-Viral or aseptic meningitis : acute febril illness

Symptoms :

1)Headache

2)Signs of meningeal irritation.

3)Petechiae or skin rash may occur in patients with enteroviral meningitis.

Prognosis:

Unless associated encephalitis (meningeoencephalitis) exists recovery is uneventful .

5-Enteroviral eruptions may occur in patients infected with either echo or coxsackieviruses . These often cause exanthematous eruptions as well as febrile illness.

- Serological profile of hepatitis A virus infection.

1)The viremic stage of infection occurs during the prodromal phase and is of a short duration and of low titer.

2)Anti HAV IgM appears during the acute phase and declines to non detectable levels within 3-6 months.

3)Anti HAV IgG rises 2-3 weeks later and gives life long protection.

- Bacteriological diagnosis of a typhoid carrier.

In search for typhoid carriers either as part of routine examination of food handlers or after an outbreak of typhoid fever .

A useful screening test is to examine blood serum specimens for specific antibodies ,in particular if the Vi antibody is present in a titre of 20 or higher the individual may be a carrier and should have several stool cultures plus bile culture after duedenal aspiration .

-Pereferably hospital patients may be similarly examined six months or more after discharge from hospital ,if there was a significant titer at the time of discharge.

- Vaccination for poliomyelitis.

Trivalent poliovaccine is generally used .Both live virus and killed virus vaccines are available :

a. Formalinized vaccine (Salk) is prepared from virus grown in human diploid cell cultures. At least four inoculations over a period of 1-2 years have been recommended in the primary series. Periodic booster immunizations have been necessary to maintain immunity. Inactivated-virus vaccine induces humoral antibodies, but, upon exposure, virus is still able to multiply in the gut.

b. Oral vaccines (Sabin) contain live attenuated virus grown in human diploid cell cultures. The vaccine can be stabilized by magnesium chloride, 1 mol/L, so that it can be kept without losing potency for a year at 4°C and for weeks at moderate room temperature (about 25 °C). Nonstabilized vaccine must be kept frozen until used.

The live poliovaccine multiplies, infects, and thus immunizes . In the process, infectious progeny of the vaccine virus are disseminated in the community .Repeat vaccinations are important to establish permanent immunity

The vaccine produces not only IgM and IgG antibodies in the blood but also secretory IgA antibodies in the intestine, which then becomes resistant to reinfection.

c. The application of recombinant DNA technology may permit the development of a live poliovirus that cannot mutate to increased neurovirulence. This is still under current investigation

2. Mention the mode of transmission of hepatitis B virus and its lab diagnosis.

There are three main modes of transmission
1-Percutaneous and permucosal exposure to blood:
- Transfusion of blood and blood products.
- Sharing of contaminated needles and syringes (parenteral drug addicts).
- The use of improperly sterilized instruments (even in tattooing and ear piercing).
- Sharing of razors and tooth brushes.
- Needle-stick injuries.
2- Sexual transmission.
3- Perinatal transmission from mother to newborn during birth or breast feeding. In-Utero transmission is rare.

3. Discuss the lab diagnosis of a case of cholera.

Stools of cholera patients contain a large number of vibrios (10^8/ml) and this makes the diagnosis easy. The stool should be collected before administration of antibiotics.

Specimen:

Faecal specimen may be obtained by:
1. Rubber catheter into a sterile container.
2. Rectal swab which should absorb about 0.1-0.2 ml.
3. Collection from a bed pan should be avoided because of the risk of contamination or the presence of antiseptics.

I. Microscopy (Rapid diagnosis):

This is done when there is urgency in making the diagnosis (during epidemics) but it should be confirmed by culture.

a) Immobilization under dark field microscopy

Two drops of fresh fluid specimen or two drops of a young peptone water culture on a glass-slide, examined by dark ground microscopy to demonstrate the motility, then a drop of either Inaba or Ogawa antiserum is added to the bacterial suspension. Specific immobilization can be demonstrated in 60-80% of acute cases of cholera.

b) Fluorescent antibody technique

A faecal smear is made on a slide and stained with the direct or indirect fluorescent antibody technique. It takes about two hours and diagnoses 90% of cases but it needs an ultra-violet microscope.

II. Culture:

1. Faecal specimens are inoculated in:

♦ A tube of alkaline peptone water incubated for 6 hrs at 37$_{o}$C then subcultured on a plate of T.C.B.S.
♦ A large loopful over a plate of TCBS medium, then incubated at 37 °C overnight.
2. Bacteria that form yellow colonies on TCBS are examined by slide agglutination with V. cholerae O$_{1}$ antiserum then with Inaba-Ogawa antisera.
3. The identity is confirmed by demonstrating motility and oxidase test.
4. The biovar may be determined by the different reactions listed in the table.

Table 8- 8: Characters differentiating the V. cholerae biovars:

Feature	**Classical**	**V cholerae El-Tor**
– Haemolysis of sheep RBCs	-	+
– Haemagglutination of chicken RBCs	-	+
– Voges-proskauer reaction	-	+
– Susceptibility to polymyxin B	Sensitive	Resistant
– Susceptibility to cholera phage IV	Sensitive	Resistant

III. Molecular methods:
1. PCR amplification has been used for the detection of the cholera toxin A subunit gene in the rice water stools.
2. DNA probe for detecting toxigenic V. cholera

4. Discuss the lab diagnosis of Helicobacter pylori infection in a patient with chronic gastritis.

Through invasive or noninvasive methods.

I. Invasive methods:
It involves gastroscopy and obtaining antral biopsy specimens. The gastric biopsies are used for:
1. Direct urease test, using urea broth or agar for detection of urease enzyme.
2. Culture on Skirrow's medium.
3. Histopathological examination to demonstrate the bacteria and to diagnose the histopathological lesion.

II. Non invasive method:
1. Urea breath test, which relies on the presence of H. pylori urease, the patient ingests radioactively labeled (^{14}C) urea, and if infection is present, the urease of H. pylori hydrolyzes the urea to form ammonia and labeled

bicarbonate that is exhaled as CO_2, the labeled CO_2 is then detected in expired air.

2. Detection of H.pylori antigens in stools by ELISA for diagnosis and to monitor treatment

5. Discuss briefly E.coli diarrhoae.

Strains of certain serotypes of E. coli have a primary pathogenicity in the intestine and cause gastroenteritis. They fall into four distinct groups associated with different disease syndromes:

1. Enteropathogenic E. coli (EPEC):

These E.coli produce bundle forming pili (Bfp) and intimin adhesine which allow bacterial attachment to epithelial cells of small intestine leading to disruption of the microvilli (an attaching-effacing mechanism of action) leading to watery diarrhea. EPEC strains belong to particular serotype sand they cause sporadic cases and outbreaks of watery diarrhea in babies and young children.

2. Enterotoxigenic E. coli (ETEC):

They are a common cause of acute watery diarrhoea in developing countries. It may affect infants, children and adults. Responsible also for about half of the episodes of diarrhoea in travelers to developing countries, (traveler's diarrhoea),they possess colonization factors (fimbrial adhesins) which bind the bacteria to specific receptors on the cell membrane of the small intestine ,also two distinct enetrotoxins are produced a heat-labile toxin (LT) and a heat-stable toxin (ST). They mediate hypersecretion of water and electrolytes into the bowel lumen.

3. Enteroinvasive E. Coli (EIEC):

Certain serotypes of E. coli have been implicated as a cause of dysentery like diarrhoea with blood, and mucus in the stools. They resemble Shigella in their pathogenicity, as they invade the enterocytes lining the large intestine.

4. Enterohemorrhagic E. coli (EHEC):

These E.coli produce verotoxin which is identical to shiga toxin .After attaching to mucosa of the large intestine, the produced toxin has a direct effect on intestinal epithelium resulting in bloody diarrhea, the organism causes hemorrhagic colitis where there is destruction of the mucosa and consequent hemorrhage, this may be followed by hemolytic uremic syndrome .Verotoxin receptors have been identified on renal epithelium and may account for kidney involvement. There are many serotypes of EHEC, the commonest is O157:H7

6. Discuss the different clinical syndromes caused by Cocksakie viruses.

Coxsackieviruses, a large subgroup of the enteroviruses, are divided into two groups, A and B, having different pathogenic potentials for mice. They produce a variety of illnesses in humans.

Clinical Syndromes:

1. *Coxsackie A:*

a. Herpangina. The classic finding is vesicular, ulcerated lesions around the soft palate and uvula. The disease is self limited and requires only symptomatic management.

b. A Hand-foot- and mouth disease is a vesicular exanthem caused by an enterovirus, usually coxsackievirus A 16,

c. Enterovirus 70 and a variant of coxsackievirus A24 have recently been associated with an extremely contagious ocular disease, acute hemorrhagic conjunctivitis. The disease has an incubation period of one day and resolves within one or two weeks.

d. Cold like symptoms.

e. Aseptic meningitis caused by many types of group A.

2. *Coxsackie B:*

a. Epidemic myalgia (Pleurodynia or Bornholm disease): also known as the ―devil's grip‖. It is an acute illness in which patients have fever and a sudden onset of agonizing stitch like pains in the muscles of the chest and

epigastrium. Abdominal pain and even vomiting may also occur. The illness is selflimited and recovery is complete, though relapses are common.

b. Myocardial and pericardial infections caused by coxsackie B virus occur in older children and adults but are most threatening in newborns. Coxackie B viruses are the most commonly identified agents of viral heart disease in humans.

c. Seroepidemiologic evidence links type I diabetes mellitus (insulin dependent) with coxsakie B viruses.

d. Viral or aseptic meningitis is an acute febrile illness accompanied by headache and signs of meningeal irritation. Petechiae or skin rash may occur in patients with enteroviral meningitis. Unless associated encephalitis (meningoencephalitis) exists recovery is uneventful.

e. Enteroviral eruptions may occur in patients infected with either echo or coxsackieviruses. These often cause exanthematous eruptions as well as febrile illness.

7. Describe the dane particle. Draw and label the serological profile of hepatitis B virus infection.

It's the 42 nm complete virions (less frequently observed).

**Refer figures pages 66 and 67

8. Enumerate clinically important vibrios and the disease they produce.

1. V. cholerae and its biovar El-Tor are the causative agents of epidemic and endemic cholera (serogroup O_1). Recently serogroup O_{139} was also found responsible for several epidemics.
2. Non-cholera vibrios which share the H antigen but not the O antigen with V. cholerae. They are associated with diarrheal illnesses, though usually much less severe than classical cholera.
3. Halophilic vibrios: These require sodium chloride for growth, e.g. V. parahaemolyticus which causes a type of food-poisoning or acute gastroenteritis following ingestion of contaminated sea food.

9. List the names of bacteria that cause toxin mediated food poisoning.

1-Staphylococcus aueus food poisoning.

2-Botulism.

3-Infant botulism.

4-Bacillus cereus food poisoning.

10. A 50-year old man has diffuse gastritis and a peptic ulcer on endoscopic examination. A biopsy specimen was obtained.

a) What is the bacteria most likely to be seen in the histopathologic exam of this biopsy?

H.pylori

b) What are the other methods performed using this specimen to confirm the diagnosis?

Through invasive or noninvasive methods.

I. Invasive methods:

It involves gastroscopy and obtaining antral biopsy specimens. The gastric biopsies are used for:
1. Direct urease test, using urea broth or agar for detection of urease enzyme.
2. Culture on Skirrow's medium.
3. Histopathological examination to demonstrate the bacteria and to diagnose the histopathological lesion.

II. Non invasive method:

1. Urea breath test, which relies on the presence of H. pylori urease, the patient ingests radioactively labeled (^{14}C) urea, and if infection is present, the urease of H. pylori hydrolyzes the urea to form ammonia and labeled bicarbonate that is exhaled as CO_2, the labeled CO_2 is then detected in expired air.

2. Detection of H.pylori antigens in stools by ELISA for diagnosis and to

monitor treatment

c) What are the main virulence factors for this organism?

The main virulence factors of the organism are the urease enzyme, the protease enzyme and the vacuolating cytotoxins which causes damage to the host cells

d) Write briefly about the urea breath test.

. Urea breath test, which relies on the presence of H. pylori urease, the patient ingests radioactively labeled (^{14}C) urea, and if infection is present, the urease of H. pylori hydrolyzes the urea to form ammonia and labeled bicarbonate that is exhaled as CO_2, the labeled CO_2 is then detected in expired air.

11. Give an account on the importance of detection of the following hepatitis A markers:

- IgM anti HAV

Confirms the diagnosis of of acute hepatitis A (The most reliable single assay)

- IgG anti HAV

Denotes past infection or vaccination

- HAV antigen in stools.

12. Discuss the non invasive methods to diagnose a case of helicobacter pylori.

1. Urea breath test, which relies on the presence of H. pylori urease, the patient ingests radioactively labeled (^{14}C) urea, and if infection is present, the urease of H. pylori hydrolyzes the urea to form ammonia and labeled bicarbonate that is exhaled as CO_2, the labeled CO_2 is then detected in expired air.
2. Detection of H.pylori antigens in stools by ELISA for diagnosis and to monitor treatment

13. Define the structural properties of HDV. Explain why it can't replicate on its own.

.

Virions consist of spherical particles 35-37 nm in diameter. HDV has small circular RNA genome. The RNA is a ss negative sense closed circle which encodes only one protein, the internal core protein HDAg or delta antigen.

*Hepatitis D virus is a defective virus, it can not replicate by itself because it does not have the genes coding for its envelope protein. HDV can replicate only in

cells infected with HBV, because HDV uses the HBsAg as its envelope protein. HBV is therefore the helper virus for HDV

14. Outline the rapid tests used to diagnose cholera in epidemics.

Microscopy (Rapid diagnosis):

This is done when there is urgency in making the diagnosis (during epidemics) but it should be confirmed by culture.

a) Immobilization under dark field microscopy

Two drops of fresh fluid specimen or two drops of a young peptone water culture on a glass-slide, examined by dark ground microscopy to demonstrate the motility, then a drop of either Inaba or Ogawa antiserum is added to the bacterial suspension. Specific immobilization can be demonstrated in 60-80% of acute cases of cholera.

b) Fluorescent antibody technique

A faecal smear is made on a slide and stained with the direct or indirect fluorescent antibody technique. It takes about two hours and diagnoses 90% of cases but it needs an ultra-violet microscope.

15. What is the significance of the detection of:

- Anti HCV

HCV infection is usually diagnosed by detecting antibodies to HCV by ELISA.

- In testing for anti-HCV the following has to be considered:

1- Negative results may be obtained in the early seronegative phase.
2- A positive test does not distinguish between acute, chronic or resolved infection.
3- False-positive results can occur in ELISA, a RIBA (recombinant immunoblot assay) should be performed as a confirmatory test. If the results of RIBA are positive, testing for the presence of viral RNA in serum should be done to determine whether active disease exists.
4- Some HCV infected patients have a poor serologic response (eg. chronic hemodialysis patients). The presence of infection can be confirmed by testing for HCV RNA.

- Anti HBe

In acute hepatitis :Denotes the start of the resolution of the disease.

In chronic hepatitis: Low propability of transmission of the disease.

- HBeAg in case of chronic HBV

High propability of transmission.

6- pharmacology

1. Mention the side effects and drug interactions of antacids.

Drug interactions: Antacids alter the bioavailability of many drugs by the following mechanisms:

1. They increase gastric PH decreases absorption of acidic drugs & increase that of basic drugs.

2. The metal ion in some preparations can chelate other drugs, e.g. digoxin, tetracycline and prevent their absorption.

3. The alkalinization of urine produced by certain antacids increases excretion of acidic drugs & decreases the excretion of basic drugs.

Side Effects :

1. **Sodium bicarbonate**

* Short duration of action.

* Systemic absorption, so prolonged use may cause systemic alkalosis & fluid retention (contraindicated in patients with salt & water retention)

* CO2 liberation that leads to re-stimulation of HCl secretion (Acid rebound), gastric distension, perforation of the ulcer & hemorrhage.

2. **Mg carbonate**

* CO2 liberation that leads to re-stimulation of HCl secretion (Acid rebound), gastric distension, perforation of the ulcer & hemorrhage.

* Laxative effect and may cause diarrhea

* Hypermagnesemia in cases with renal impairment.

3. **Mg oxide and hydroxide**

* Laxative effect and may cause diarrhea

* Hypermagnesemia in cases with renal impairment.

4. **Mg trisilicate**

* Slow onset of action

* Laxative effect in large dose

* Hypermagnesemia in renal impairment.

5. **Aluminum hydroxide**

* Slow onset of action.

* Constipating effect.

* Al+3 ions form complexes with certain drugs thus decreasing their bioavailability (e.g. tetracyclines)

* Hypophosphatemia

2. Give an example for the first line regimen used for helicobacter pylori eradication and mention its efficacy and disadvantages.

PPI-based three drug regimen:

this is commonly employed as a 1st line regimen.

It consists of a combination of any PPI and two of the following three antimicrobial agents: Clarithromycin, Amoxicillin or Metronidazole,

given twice daily for at least 7 days,

preferably 10-14 days

Side effects of H. Pylori eradication therapy:

1. Treatment failure

2. Antimicrobial agents for H pylori can increase other organism resistance as Strept. pneumonia.

3. Nausea, vomiting and abdominal cramps

4. Diarrhea: 30-50% of patients but usually mild

5. Metronidazole causes metallic taste (common)

6. Headache

7. Rash

3. Write short notes on omeprazole.

أحنا فاضين D:حنكتب الصفحة و نص كلهم بقا

الأسئلة إن شاء الله تيجي أسهل من كدا ، زي اللي فات ، متقلقوش ، بس اقروه للمراجعة كدا :)

Omeprazole is proton-pump inhibitors (PPIs), irreversibly inhibits H+/K+ ATPase enzyme system suppressing secretion of hydrogen ions into the gastric lumen.

They inhibit more than 90% of both basal and stimulated gastric acid secretion.

Pharmacokinetics:

provided as enteric-coated preparations to protect them from premature activation by gastric acid.

After absorption in the duodenum, they are transported to the acid parietal cell canaliculus, where they are concentrated by ionic trapping and converted to active form.

The bioavailability of all agents is decreased approximately 50% by food; hence, the drugs should be administered on an empty stomach.

Since PPIs inhibit only Na/K ATPase that are actively secreting acid, they should be administered approximately 30-60 min before a meal (usually breakfast), so that the peak serum concentration coincides with the maximal activity of proton pump secretion.

The drugs have a short serum half-life of about 1.5 hours, but acid inhibition lasts up to 24 hours owing to the irreversible inactivation of the proton pump. PPIs are metabolized by the liver and need dose adjustment in severe hepatic impairment.

Therapeutic Uses:

1. Peptic ulcer

- Healing of ulcer (conventional therapy)
- Prophylaxis against NSAIDs induced ulcer
- Part of H.pylori eradication regimen

2. Gastroesophageal reflux disease (GERD) especially erosive esophagitis.

3. Zollinger-Ellison syndrome and other hypersecretory states.

4. Prevention of stress related mucosal bleeding in critically ill patients (Patients in intensive care units).

Adverse Effects:

1. Headache, diarrhea and rash.

2. Increased concentration of viable bacteria in the stomach.

Drug interactions:

1. Omeprazole is an enzyme inhibitor that interferes with the metabolism of warfarin, phenytoin, diazepam and cyclosporine.

2. Because all PPIs increase intragastric pH, they may alter the bioavailability of some orally administered drugs, such as ketoconazole (weak bases), or in pH-dependent dosage forms.

4. Give examples for combination therapy of antiemetics

* Aprepitant combined with 5HT3-receptor antagonist and Dexamethasone is the standard antiemetic therapy for prevention of nausea and vomiting induced by highly emetogenic chemotherapy

* Corticosteroids, most commonly dexamethasone, increase antiemetic activity when given with high dose of metoclopramide, a 5HT3 antagonist, phenothiazines, butyrophenones, a cannabiniods or benzodiazepines

* Antihistaminics like diphenhydramine + high dose of metoclopramide reduce the extrapyamidal side effects of metoclopramide

* Corticosteroids + metoclopramide reduce the metoclopramide induced diarrhea.

5. Name an antiemetic used during pregnancy and its mechanism of action.

Doylamine ,Histamine-1 (H1) -receptor antagonist

most likely act by inhibiting cholinergic pathways of the vestibular apparatus. It appears probable that the significant antimuscarinic and sedative effects and the H1 blocking effect contribute to their anti- emetic efficacy.

H1-receptor antagonists are used to treat motion sickness, true vertigo, and nausea & vomiting of pregnancy.

6. For peptic ulcer treatment of a patient with hepatic impairement, which is preferred, ranitidine or cimetidine? Explain why.

Ranitidine

it's well tolerated and may have only the minor side effects observed with cimetidine. They have neither C.N.S nor anti-androgenic effects.

Cimetidine has drug interactions inclding :

Microsomal enzyme inhibition: cimetidine inhibits cytochrome P-450 and so can slow the metabolism and thus potentiate the action of several drugs e.g. warfarin, diazepam, phenytoin, quinidine, theophylline, imipramine...etc. Ranitidine interferes only minimally with hepatic metabolism of other drugs. Famotidine and nizatidine are even safer with no significant drug interactions mediated by inhibiting hepatic CYPs.

7- Biochemistry

1) Discuss in brief Lactose intolerance . (mention cause and pathogenesis) . (July 2014)

- Cause : lactase deficiency . due to :

1. Genetic defect or 2. Injury to mucosa (intestinal disease or drugs) or 3. Physiologic decline with age.

- Pathogenesis :

Lactase deficieny >> passage of undigested lactose into large intestine >> (Osmotically active material) so draw up water from mucosa into large intestine >> Osmotic diarrhea .

This is reinforced (+++) by action of intestinal bacteria (Fermentation) of the remaining carbohydrates to two and three compounds (osmotically active , as well)

+ large amounts of CO2 and gas >> Faltulence

---

2) Illustrate different mechanisms that are common between Glucose and amino acids absorption . (July 2013)

Glucose absorption

- Carrier mediated process .
- 2 systems :
  1) Na dependant carrier ptn (found on the brush border) : Requires uptake of sodium and D Galactose and D Glucose . (needs energy provided indirectly , by the active transport of Na+ out of the cell (Na+ K+ ATPase pump) . This maintain concentration gradient across luminal border .
  2) Na independent carrier ptn :
     -for D-Fructose and pentose
     - with concentration gradient (so remove it by its transport ;)
     - Doesn't need energy .

Protein absorption

- End products are a.a & di and tri peptides
- They are absorbed via amino acid or peptide transport system .
- It is an active process against concentration gradient .. it needs Na+ as co-transport system from luminal surface . ATP is the source of energy .
- After absorption di and tri peptides complete digestion to free a.a

---

3) Give a short account on :

1. **Hartnup's disease .**

- It is a Genetic disease characterized by inability of renal tubules and intestinal cells to transport neutral amino acids > amino aciduria
- This will lead to manifestation of deficiency of essential amino acids (amino acids which cannot be synthesized inside the body) eg : Tryptophan .

2. **Absorption of monosaccahrides .** (Oct, 2014)

Answered before .

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4)Discuss the digestion of phospholipids . (mention name of enzyme , mechanism of action and products). (June, 2013)

- **In Mouth :**

No digestion of lipids .

- **In Stomach** :

Digestion occurs in children by enzyme Gastric lipase .

It's effective only in children due to :

1) High Ph in their stomach

2) And their milk diet with short chain fatty acids .

- **In intestine :**

Didestion of lipids occur mainly by pancreatic and intestinal enzymes .

1)Pancreatic lipase : major enzyme of TGs hydrolysis (ph =7)

- Mechanism of action :

1) Emulsification is done by (mechanical agitation in Gut + bile salts + co-lipase + phospholipids).
2) This enzyme is specific for TGs esters in alpha position (linkage between 1 and 3 of TG).
3) Prefers long chain fatty acids .

- End products :

1) 2-mono acyl glycerol (72%)
2) 1-mono acyl glycerol (6%)
3) Completely hydrolyzed to free fatty acids FFA (22%).

2)Cholesterol ester hydrolase (found in pancreatic juice) :

- Mechanism of action :

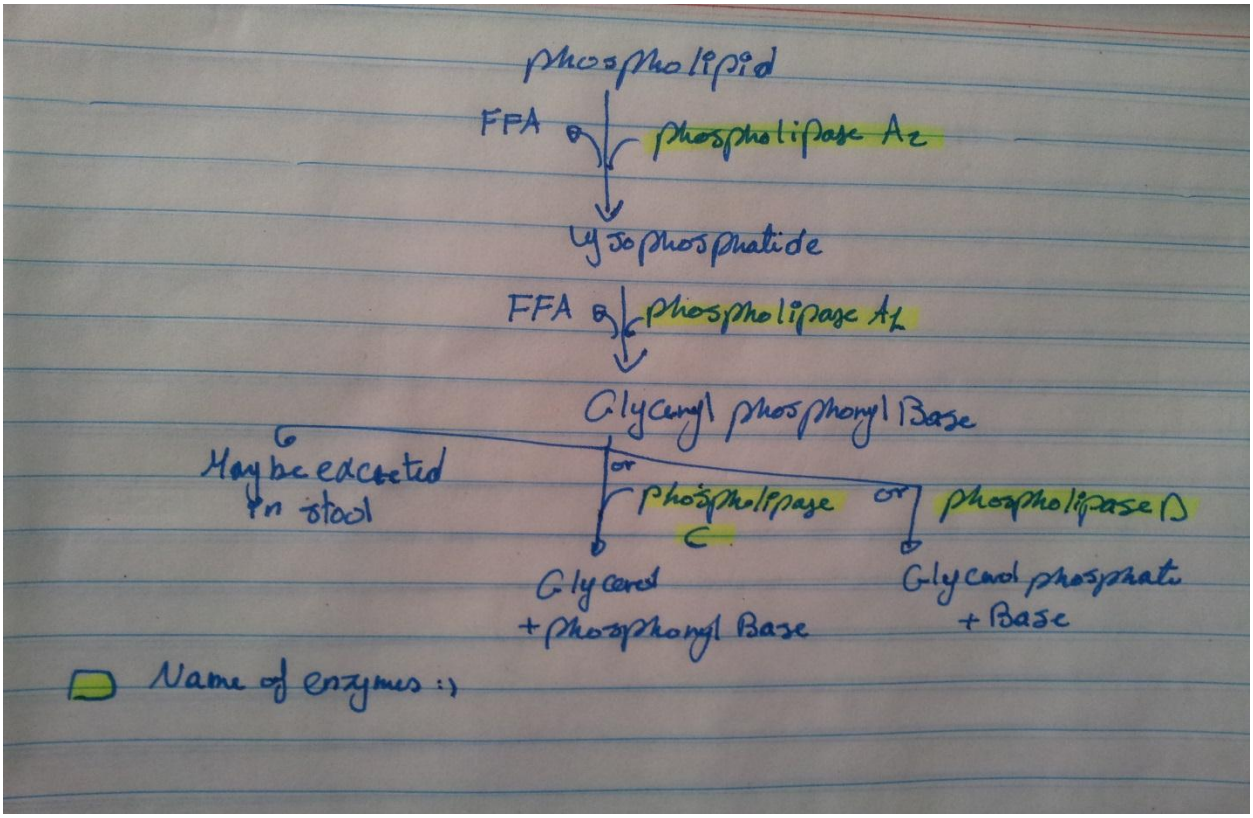
it catalyzes hydrolysis of cholesterol esters .

- End products :

FFA.

3)Phspholipase A2 and other phospholipases :
[Phspholipase A1 , Phspho Lipase C , and Phspholipase D]

- Mechanism of action :



- End products :

1) Lysophosphatide (mainly) .
2) FFA .
3) Glycerol phosphate .
4) Nitrogenous base .